VOLUME XXII NUMBER 1

DISEASES

of the

CHEST

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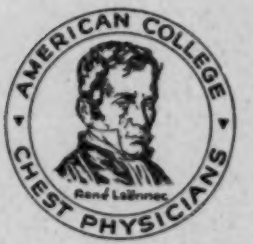

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Rio de Janeiro, Brazil, August 24-30, 1952

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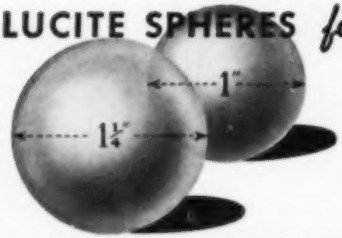

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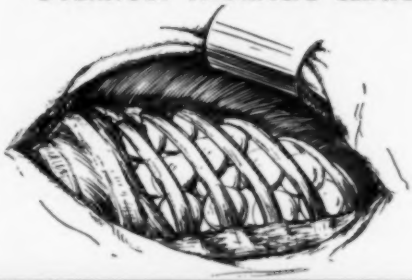

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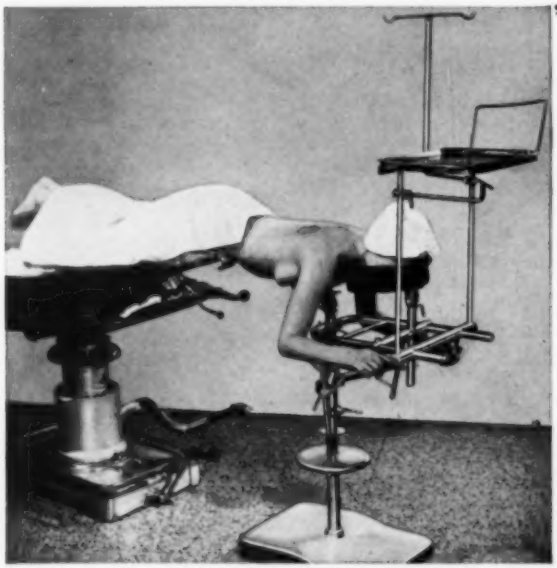

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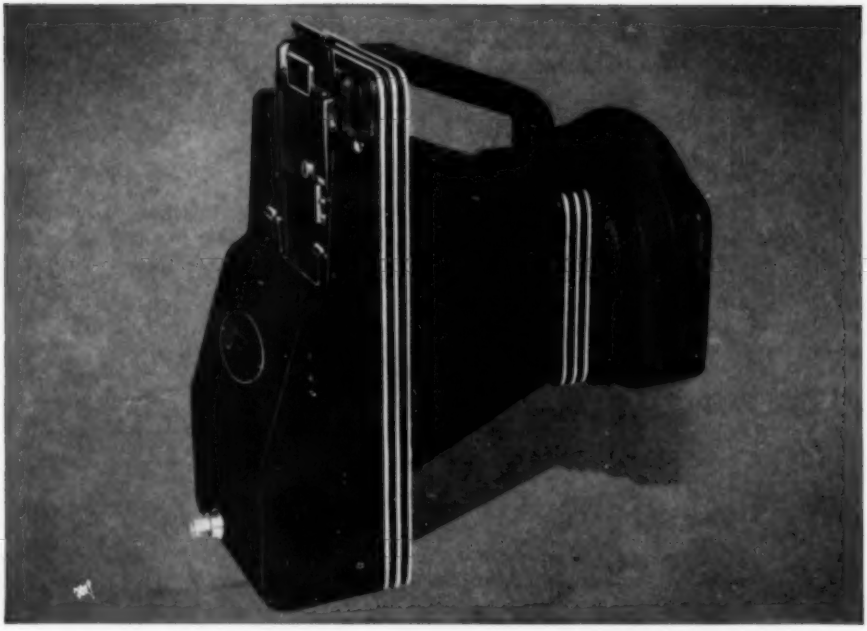

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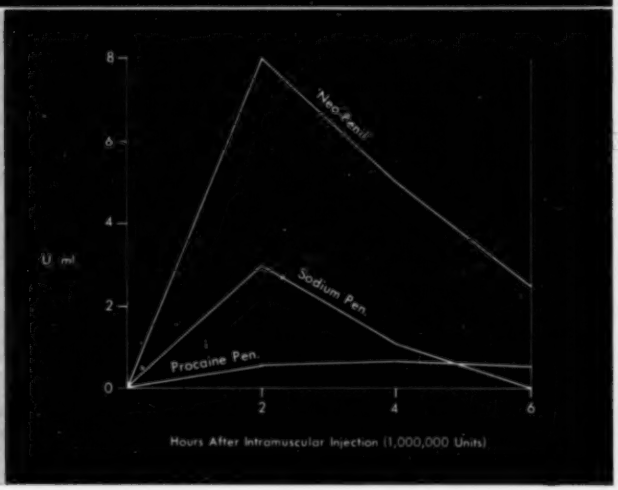

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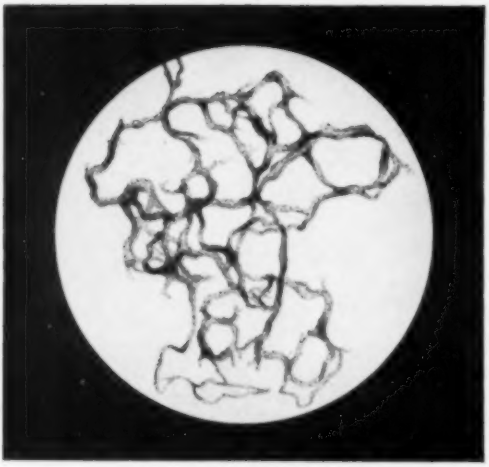

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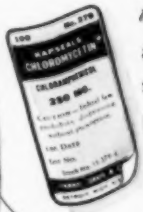

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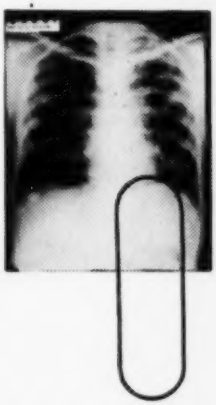

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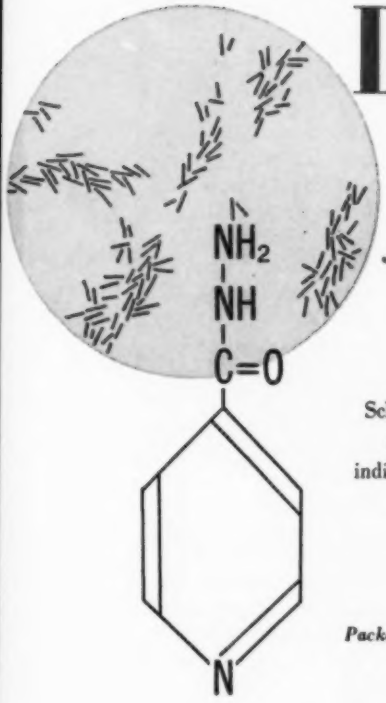

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VOLUME XXII JULY 1952 NUMBER 1

Closed Intracardiac Tactile Surgery*

C. P. BAILEY, M.D., F.C.C.P., R. P. GLOVER, M.D., F.C.C.P. and T. J. E. O'NEILL, M.D., F.C.C.P.
Philadelphia, Pennsylvania

From the beginning of medical history, surgery of the heart has been a phantasy, sometimes appearing to be almost within grasp, but always slipping away behind seemingly insurmountable obstacles. While many surgeons have dared to dream, the very greatest of them have at times faltered and have erected additional mental hazards to block medical progress. Thus, pericardial effusions have been treated by aspiration or thoracostomy since the Hippocratic era. Yet even the great Bilroth in 1883 stated that "Any surgeon wishing to preserve the respect of his colleagues should never attempt to suture a heart wound" (quoted by Cutler, Levine and Beck[14]).

In 1898, Rehn[1] performed the first successful suturing of a stab wound of the heart. This is the first historical example of intrinsic myocardial surgery. This great accomplishment was followed by many similar operations. Also in 1898 Delorme[2] suggested the deliberate removal of the thickened pericardium in cases of constrictive pericarditis. This may be considered actually as pericardial surgery.

Brauer[3] suggested and first performed his "cardiolysis" for great enlargement of the heart. This was not actually a cardiac operation, consisting as it did of removal of segments of the overlying ribs and other rigid precordial tissues to decompress the restricted myocardium.

*Presented at the New York State Chapter Meeting, American College of Chest Physicians, Buffalo, May 3, 1951, and the California Chapter Meeting, American College of Chest Physicians, Los Angeles, May 8, 1951. First Annual Howard Lillienthal Lecture at New York State Chapter Meeting.

From the Department of Thoracic Surgery, Hahnemann Medical College and Hospital of Philadelphia, Pennsylvania.

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The great accomplishments of Graybiel, Strieder, and Boyer[4] and Gross[5] in the obliteration of patent ductus arteriosus, and of Craaford[6] and Gross[7] in excising the stricture in aortic coarctation with subsequent end-to-end anastomosis were epochal in nature. However, they were not performed for any inherent or intrinsic cardiac defect or deformity and may properly be considered as *major vascular surgery*.

The dramatic systemic artery—pulmonary artery anastomosis of Blalock and Taussig[8] and the equally important aortic-pulmonary arterial shunt of Potts, Smith and Gibson[9] were performed for intrinsic cardiac deformity (Tetralogy of Fallot). They were not, however, actually operations upon the heart itself, but upon the great vessels arising from it. We may therefore consider these procedures as *major cardiovascular surgery*. However, Doyen[10] in 1913 had already performed the first known intracardiac operation in a human heart, a transventricular incision of the pulmonary valve, for pulmonary stenosis. This was followed by the brilliant but largely tragic efforts of the great pioneers of mitral surgery, Cutler and Levine,[11] Pribram,[12] Allen and Graham,[13] and Cutler, Levine and Beck.[14] The only notable success in this period was that of Souttar[15] who digitally dilated a stenotic mitral valve via the left auricular appendage. This was undoubtedly the first commissurotomy, by the finger method.

It was not until June 1948 that another successful mitral commissurotomy was accomplished, this one by Bailey[16] by the incisional method. The knife was guided by the index finger to which it was bound by a rubber sheath. The finger again approached the mitral valve by way of the left auricle and its auricular appendage. The subsequent success of this principle of commissurotomy, either by the intracardiac finger alone (finger pressure to split the valve) or by the knife guided by the intracardiac finger, has rendered obsolete, contemporary efforts at other types of valvulotomy by Smithy,[17] Harken, Ellis, Ware, and Norman,[18] and Murray.[19] The azygos vein—pulmonary vein shunt of Sweet and Bland[20] has also become less useful with the improvement in the technique and results of the direct valvular attack.

In 1948, Brock[21] performed his first successful surgical division of a stenotic pulmonary valve, entering the valve by an incision in the wall of the right ventricle. Subsequently he developed his technique of punching out a window in the obstructing infundibular septum in tetralogy of Fallot. This technique has since been extended and modified by him and by Sellors,[22] and by Blalock and Kieffer.[23] Subsequently, Downing, Bailey and Glover,[24] and Glover, Bailey and O'Neill[25] have extended it to include not

only division and dilatation of the stenotic pulmonary valve, but also dilatation of the strictured main pulmonary artery and partial rongeur resection of an obstructing infundibular septum. These latter cases usually presented the classical picture of Tetralogy of Fallot, a condition formerly always treated by either the Blalock or the Potts type of shunt. Should the technical problems and the higher initial operative mortality associated with this direct attack in pulmonary stenosis become somewhat lessened, the shunt operations will no doubt become second choice procedures to be employed chiefly when the direct operation is for some reason contra-indicated.

Since the palpating finger is being more and more employed within the beating right ventricle to locate and diagnose the obstructing lesion, to guide the attached cutting knife, or to dilate the partially divided obstruction, this modified Brock procedure may, like modern mitral commissurotomy, be properly considered as a prime example of closed intracardiac tactile surgery.

Closed intracardiac tactile surgery differs as much from standard surgical techniques as does endoscopic surgery (bronchoscopy, closed pneumonolysis, transurethral resection, etc.), or manipulative orthopedic surgery. Yet it is just as rational and undoubtedly for its purposes, will eventually prove just as completely acceptable. It embraces the principle of operating within the chambers of the beating functioning heart, usually using an intracardiac finger either to actually perform the pertinent manipulations, or to guide an instrument in its movements within the heart, or both. The finger must be introduced into the heart in such a way that it does not seriously hamper the cardiac action, at a point in the cardiovascular pumping mechanism where adequate temporary hemostatic control is easily accomplished, and where eventual satisfactory and secure suturing is feasible. The instruments used for this work must be either rounded or of very small calibre, so that they may be introduced through the heart wall without producing serious bleeding, or they may be flattened to be passed into the heart along with the exploring finger, bound to it by a rubber sheath. However, in a few types of cases, it is more logical to place the instrument within the heart, and the guiding palpatory finger upon the surface of the heart or adjacent great vessels rather than actually within the chambers. Tactile vision is employed in this type of operative surgery, rather than the clear, direct ocular vision of the definitive operative field upon which many surgeons so strongly insist, or the clear ocular telescopic vision upon which the endoscopist depends. It is the "vision" of the blind man, which enables him to perform the most intricate and complicated maneuvers in his world of darkness. For certain

FIGURE 1

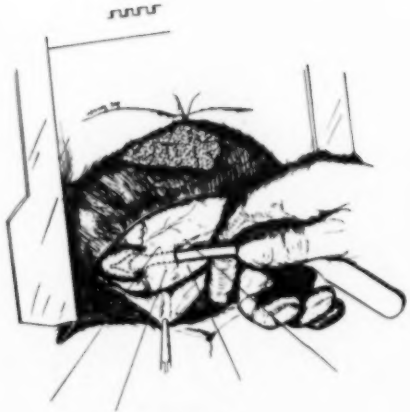


FIGURE 2

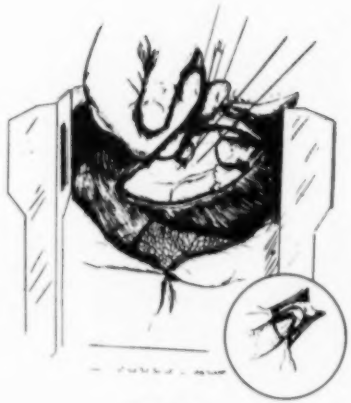


FIGURE 3

Figure 1: Digital dilatation or fracture of stenotic tricuspid valve.—*Figure 2:* Brock valvulotomy for valvular pulmonic stenosis.—*Figure 3:* Digital examination of the interior of the right ventricle for Tetrallogy of Fallot or pulmonic stenosis.

highly specialized scientific and industrial procedures, skilled blind operators are more valuable than any normal workman or machine. It is not suggested that only blind surgeons be trained for the performance of closed intracardiac surgery. The necessary intracardiac maneuvers are essentially simple and can well be learned by any otherwise competent thoracic surgeon. The necessary movements and degree of accuracy required are no higher than that exhibited by most amateur photographers who readily learn to load and unload light sensitive panchromatic film in absolute darkness. One must learn to develop the latent ability to use what John Deaver[26] called "the eyes on the tips of the fingers."

One may well question the necessity of such surgery, guided only by the finger. Why not use a cardioscope to see the lesion and the operative instrument? Why not make use of one of the various types of extracorporeal circulation now approaching perfection? Then wide open surgery might be performed upon the bloodless quiescent heart during a period of total diversion of the blood stream. Theoretically, both objections are valid. In practice, we fear that neither method will prove extremely practical for this generation of mankind. Cardioscopes have been devised periodically, at least since 1925 when Allen and Graham[13] first tried it in a clinical case of mitral stenosis. Murray,[27] and Bloomberg[28] and others have recently enlarged the possible application. All these telescopic mechanisms, employed as they are, in an opaque medium must utilize the principle of displacement of the blood by a transparent fluid (saline) either free or contained within a transparent membrane. The possible serious

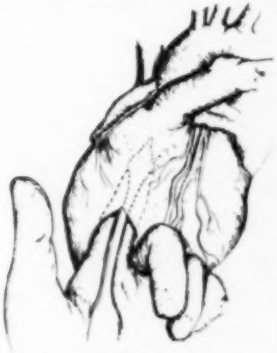


FIGURE 4

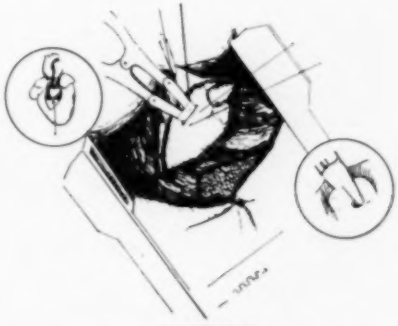


FIGURE 5

Figure 4: Passing universal valvulotome along intracardiac finger to enlarge opening in infundibular septum.—*Figure 5:* Resection of portion of infundibular septum by Glover rongeurs.

FIGURE 6a

FIGURE 6b

FIGURE 7

Figures 6a and 6b: Examination of the mitral valve and attempted "fracture" by the intracardiac finger.
Figure 7: Incision of valve commissures by guillotine knife in mitral stenosis.

interference with the flow of the circulation which such mechanisms inherently offer, the technical difficulties encountered in their employment, and in the necessary operative maneuvers, and the natural disinclination of surgeons to accept such complicated methods, bodes ill for the wide-spread acceptance of the cardioscope.

On the other hand, the extra-corporeal circulation, probably in the form of a mechanism for complete replacement of both heart and lungs is nearly upon us. Already numerous investigators including your authors[29] have developed equipment permitting complete diversion of the circulating blood in animals for an hour or more. Dennis[30] has already employed such a method in an attempt to surgically correct a human cardiac septal defect.

The great problems of satisfactory oxygenation of the blood, of pumping it without producing hemolysis or foaming, and of connecting and disconnecting the great vessels and the equipment, are well on their way to complete solution. The management of the state of coagulability or incoagulability of the blood leaves much to be desired.

The problem of maintaining coronary blood flow and of returning such blood to the circulation is a major one. It is readily apparent to the authors that even when the extra-corporeal circulation is eventually perfected, that its employment with all of the associated complications and vagaries of machinery and man

FIGURE 8a

FIGURE 8b

Figures 8a and 8b: Preparation of pedicled pericardial graft.

will be fraught with inherently greater risks than will the actual operative procedures themselves.

Consider the combined risks of connecting up the apparatus to the venae cavae and a great artery, the complete diversion of venous blood from the heart, rendering of the blood completely incoagulable, major incision into the wall of the heart, either exclusion of the arterial blood from the coronaries, or constant aspiration of excessive bleeding from the myocardial incision, the definitive operative repair, suture of the heart, restoration of a satisfactory beat, removal of the canullae, re-establishment of the normal circulation, and then restoration of normal blood coagulability!

Consider, on the other hand, that the vast majority of intracardiac defects (valvular and septal) can be readily corrected by rather simple accurate maneuvers adequately guided by the finger strategically inserted into the beating heart! It must be evident that the extracorporeal circulation and the open method of intracardiac surgery will only prove acceptable in the residual minority of conditions completely unsuitable for treatment by the closed method.

And indeed, what conditions are presently suitable for correction by closed intracardiac tactile surgery? What others may we expect will eventually prove amenable to such a method?

At the present time the authors are prepared to present suc-

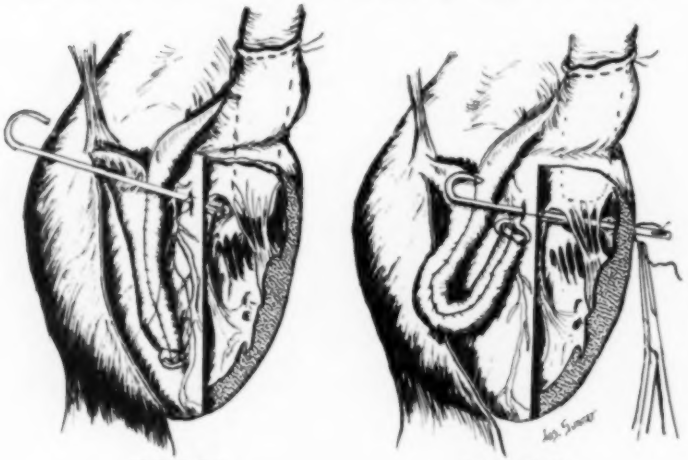


FIGURE 9a FIGURE 9b

Figures 9a and 9b: Probe is inserted through left ventricular wall and is guided by the intracardiac finger along the mitral slit and out through the posterior ventricular wall.

cessful intracardiac digital intervention in seven types of cardiac disorder, an unsuccessful attempt in another condition with a suggested improvement in technique, and three other conditions in which such intervention will probably soon be successful (based upon theoretical considerations and upon successful animal experiments).

1) *Rheumatic Stenosis of the Tricuspid Valve.*

While we have not actually cut or dilated the tricispid valve in such cases, Harken[31] has already done this in three instances. In each case the finger was inserted through the right auricular appendage into the auricle for palpation and digital enlargement of the stenotic valve orifice in a maneuver similar to his present management of mitral stenosis (Figure 1).

2) *Congenital Stenosis of the Pulmonary Valve,* or infundibular septum, with or without the associated features of Tetralogy of Fallot.

While a modification of the Brock valvulotomy (Figure 2) is used in many of the cases of valvular stenosis in our clinic, the finger is usually inserted through an incision in the wall of the ventricle to locate the obstruction in at least the doubtful cases, and to dilate most of them after the valve is incised. In cases of infundibular stenosis, where the initial incision in the myocardial wall is not located directly over the obstruction, the finger of the left hand best suited to the size of the heart (little finger for

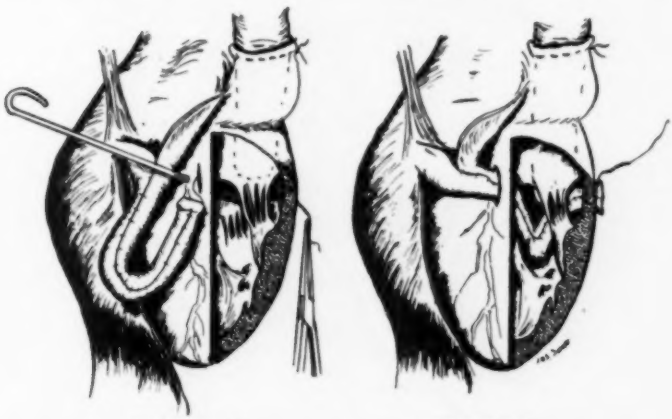


FIGURE 10a FIGURE 10b

Figures 10a and 10b: Pericardial graft is applied through left ventricular chamber in close approximation to the mitral valve.

FIGURE 11a

FIGURE 11b

FIGURE 12

Figure 11a: Lax graft floats away from mitral valve orifice during ventricular diastole.—*Figure 11b:* Lax graft is forced into the mitral orifice during ventricular systole—like a ball-valve.—*Figure 12:* Digital dilatation of stenotic aortic valve. We consider this technique very dangerous.

small children, index finger for larger children and adults) is inserted through the incision into the chamber of the right ventricle and is directed first toward the pulmonary valve and then toward the diaphragm (Figure 3). The obstructing septum must soon be encountered, and the diminutive window between the infundibular and the true right ventricular chamber is located. In some cases, a universal valvulatome (modified Brock type with flattened malleable handle) is passed along the finger to divide the obstruction or enlarge the opening in it (Figure 4). In most cases, having determined the necessary direction of approach, the finger is withdrawn from the heart and the Glover rongeur is inserted in the proper manner to grasp and resect a portion of the septum, thus producing a much larger window between the two chambers (Figure 5). The finger may then be re-inserted into the heart to determine what has been accomplished. The previous maneuvers may then be repeated if necessary.

To date we have operated upon 32 cases of Tetralogy of Fallot and six of "pure" pulmonic stenosis, using the direct (Brock) approach. In 14 of the former and one of the latter group, the finger was inserted into the right ventricle to diagnose, to dilate, or to guide the knife or rongeur. There have been seven deaths in this entire series.

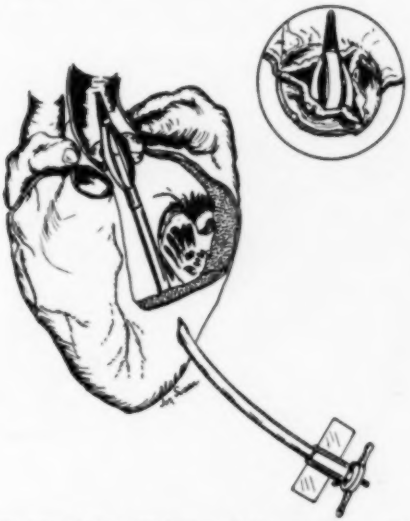


FIGURE 13: Mechanical dilatation of the stenotic aortic valve. We find this is an essentially satisfactory technique which separates one or more of the fused valve commissures without injuring the cusps or producing regurgitation.

3) *Rheumatic Stenosis of the Mitral Valve.*

This has been our surgical indication in the largest number of cases of closed intracardiac digital surgery, up to the present time. Over 200 cases of mitral stenosis, with an over-all mortality of approximately 10 per cent, have been operated on by us. Commissurotomy by either simple finger pressure or by incisional valvulotomy has been accomplished. This technique has been described in more detail elsewhere.[32-34]

Briefly, it requires insertion of the right index finger into the left auricle through an incision in the tip of the auricular appendage. The valve is palpated, the size of its orifice, thickening of its substance, flexibility, presence or absence of calcification, and presence or absence of regurgitation are estimated. The orifice may then under certain conditions, be adequately enlarged by simple digital pressure against the anterolateral commissure (Figure 6). This will, in many cases, produce a separation of the commissure to the wall of the ventricle itself. In the majority of instances, however, we feel that instrumental incision of one or both commissures is necessary. Otherwise, inadequate enlargement of the valve orifice is usual. When an instrument is used, it is so flattened that it may be inserted into the heart along with the right index finger to which it is securely but with flexibility, attached; being worn between two gloves. The left hand manipulates the

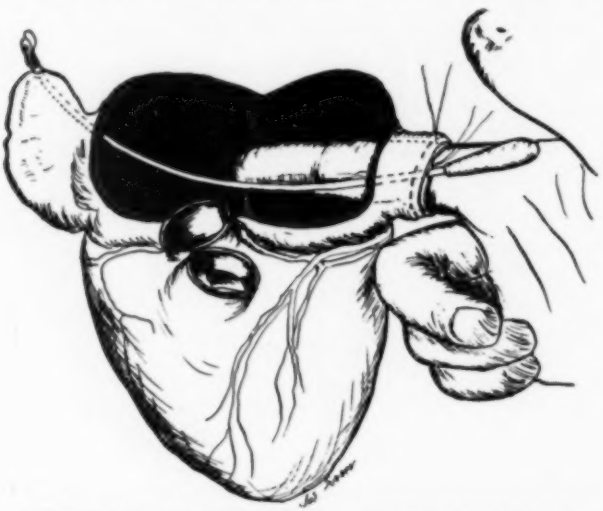
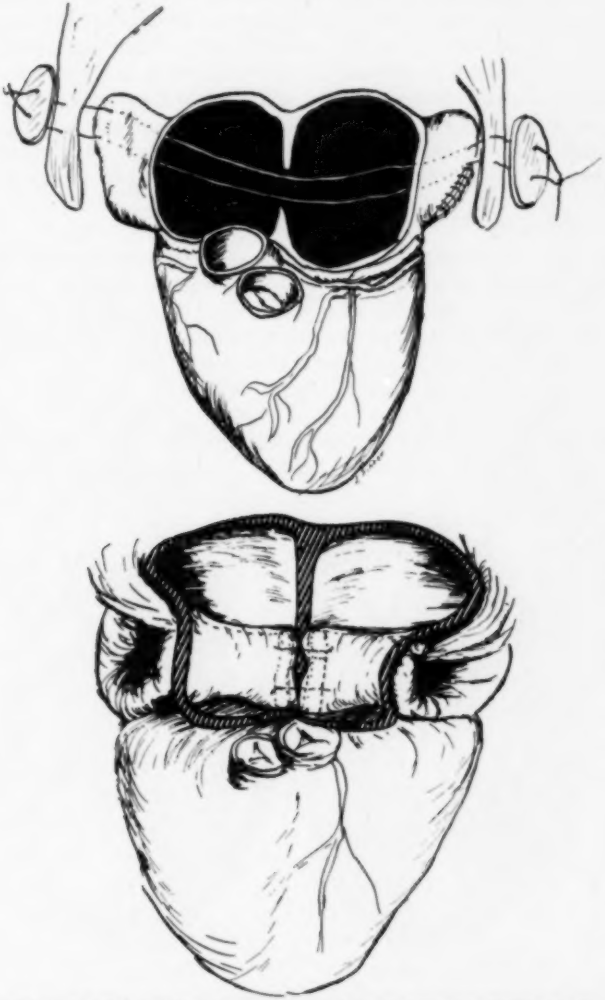


FIGURE 14: Digital localization of interatrial septal defect and accurate guidance of threaded probe through it.

handle of the instrument while the intracardiac finger of the right hand guides the cutting edge (Figure 7).

After partial section of the valve, the intracardiac finger explores and re-evaluates the enlarged orifice to determine whether



FIGURES 15a and 15b: Invagination of auricular appendages by the use of buttons of bone to approximate them within the interatrial defect with presumptive complete obliteration.

further section is desirable and to appreciate whether the pre-existing mitral regurgitation, if any, has been increased. In some cases, pressure against the partially divided valve commissure which had previously been completely resistant to the finger is able to extend and increase the valvular incision all the way to the myocardial wall, now that the heavily scarred edge has been divided. If sufficient valvular enlargement has not been produced by division of the anterolateral commissure, it is necessary also to divide the posterior one, a somewhat more dangerous maneuver. Digital splitting of this posterior commissure can only rarely be accomplished because the valve structure is not as rigidly attached to the heart wall in this region, making blunt pressure less effective.

4) *Surgical Correction of Mitral Insufficiency.*

We have operated upon 20 cases with clinical predominant mitral regurgitation confirmed at exploration. Several of these also presented a varying amount of clinically important mitral stenosis requiring a simultaneous commissurotomy. It was interesting to discover that in somewhat more than half of these cases, the valve was thickened and rigid with some over-all reduction in the size of the orifice which, however, was unable to completely close. The others presented little or no palpable thickening of the valve substance. In fact, the leaflets seemed longer and more redundant than ordinarily. The entire mitral ring in these cases appeared to be greatly dilated so that the valve edges

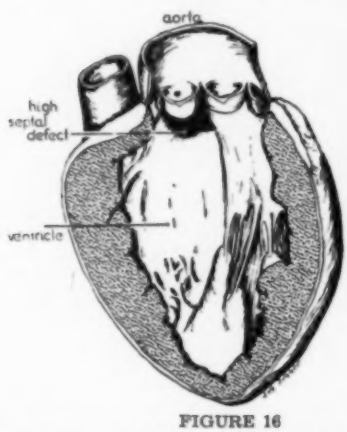


FIGURE 16

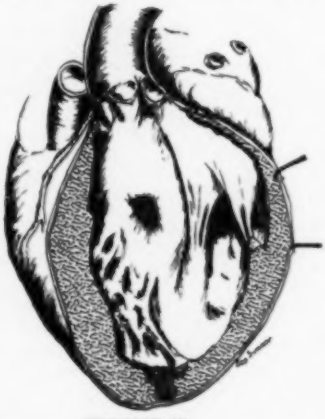


FIGURE 17

Figure 16: High ventricular septal defect as seen from the left. Note that the aortic valve makes up the top edge of the defect.—*Figure 17:* Low ventricular septal defect. The muscular septum contracts during systole lessening the amount of leak.

did not approximate upon systole, and indeed seemed to move in a paradoxical fashion. Thus, on systole of the ventricle the leaflets became separated farther than during the rest of the cardiac cycle. It appeared that the greatly dilated left ventricle abnormally separating the attachments of the papillary muscles caused them to actually pull the valve leaflets apart during systole, rather than the reverse.

This differentiation of the two types of serious mitral regurgitation is obviously of great importance since the former condition can be satisfactorily treated by application of a pedicled flap of pericardial tissue through the left ventricle just to the ventricular side of the mitral orifice. This acts as a flap valve intermittently occluding the orifice during the phase of ventricular systole. However, in cases of "valvular dysfunction" as we have called the latter condition, the size of flap capable of blocking the greatly enlarged mitral orifice would be completely unacceptable for transcardiac application. In some cases it would be more than an inch wide. Obviously some other solution must be sought.

FIGURE 18a

FIGURE 18b

Figure 18a: Preparation of tapering pedicled pericardial graft.—*Figure 18b:* Digital localization of interventricular septal defect and guidance of threaded probe through it. Continuation of insertion of curved probe brings its tip out through anterior wall of left ventricle.

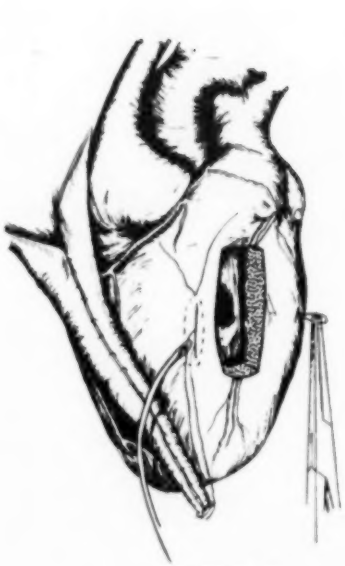


FIGURE 19a

FIGURE 19b

FIGURE 20

Figure 19a and 19b: After disengaging the probe from the suture, traction upon the latter draws the graft into the heart and through the defect.—*Figure 20:* Graft occludes the septal opening like a cork and emerges from the anterior surface of the left ventricle.

The technique of applying transventricular pericardial tamponage is essentially simple. The right index finger is inserted into the left auricle by way of the appendage and the valve is examined. If appreciable stenosis co-exists, commissurotomy is immediately performed. The pericardial graft is then prepared, with preservation of a broad attachment at the base of the pericardium to the right of the left phrenic nerve (Figure 8). Whereas formerly we merely extended the length of the graft to the diaphragm, we now, thanks to the suggestion of Victor Satinsky[35] take a spiral strip of pericardium which runs under the left phrenic nerve (without including or injuring the nerve) giving an extra 5 to 8 centimeters of length to the graft. The graft is rolled into a tubular structure with the epicardial surface on the outside. Fine interrupted arterial silk sutures maintain this rolled effect. The free tip of the graft is securely attached by a long heavy silk suture to a special probe. The probe is then inserted through the left ventricular wall just over the anterolateral commissure, as determined by the intracardiac finger. The tip of the probe soon becomes palpable to the tip of the finger inserted through the valve into the ventricle. The probe is guided by the finger past the posterior commissure and out through the posterior ventricular wall. The suture is grasped and cut free of the probe by an

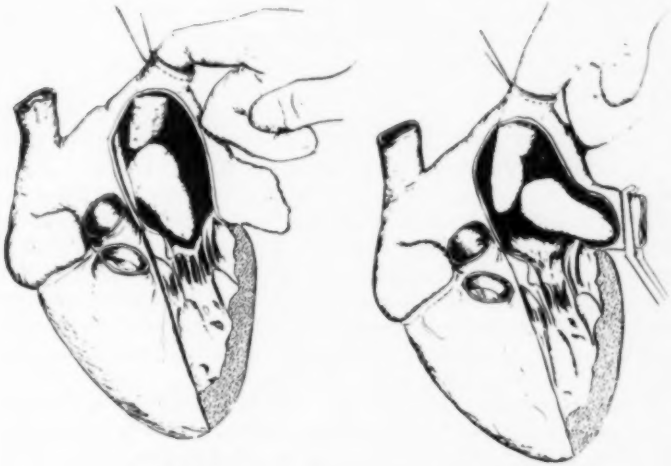


FIGURE 21a FIGURE 21b

Figure 21a: Digital dissection and mobilization of benign intra-auricular tumor arising from the septum. Finger is inserted through left superior pulmonary vein.—*Figure 21b:* Expression of mobilized tumor through incision in tip of anterior appendage.

associate. The probe is then withdrawn, and the graft is pulled completely through the left ventricle by traction upon the emerging suture. When about one and one-half inches of graft emerges from the posterior wall of the heart, the basal part of the graft is securely sutured to the anterior epicardium. The free end of the graft is then drawn back into the ventricular chamber by traction upon a heavy suture incorporated within its substance during its preparation (Larzelere suture). When sufficient slack or laxity of the graft has been provided by this maneuver, the efficient functioning of the graft can be appreciated by the intracardiac finger (Figures 10a and 10b). A graft of adequate size, properly placed, and sufficiently lax, should immediately correct 100 per cent of the regurgitation palpated with each ventricular systole (Figures 11a and 11b).

However, a graft can correct only a portion of the regurgitation in cases of "valvular dysfunction." We have been only partially satisfied by the results in such cases. For them we have devised another surgical technique whereby a suture is passed by a curved probe, completely around the chordae tendinae at the valvular orifice. This suture is then tightened to produce a "purse string-like" effect of constricting the mitral orifice. It is preferable to replace the suture with a heavy strip of fascia later for a more permanent effect. While this technique has only been applied so far in one human case, it seems effective and completely logical. The intracardiac finger again guides the maneuvers of the probe and measures the effectiveness of the procedure in correcting the regurgitation.

5) *Aortic Stenosis Can be Surgically Attacked.*

By inserting the finger through the anterior wall of the left ventricle to palpate the valve and to guide a flat knife into and through the valve orifice aortic stenosis can be surgically attacked (Figure 12). However, the inherent dangers associated with the insertion of a finger through the left ventricular wall and its subsequent maneuvers within the left ventricle are such as to militate against the wide-spread employment of such technique. Any temporary additional disturbance to its already somewhat depressed function may well prove fatal.

Serious ventricular arrhythmias are more likely to occur with irritation of the left ventricle than with similar manipulation of the right. And after performance of the definitive maneuvers, hemostatic closure of a large incision in the left ventricular wall in a case of aortic stenosis may be impossible. The severely elevated intraventricular pressure and the frequently associated very soft myocardium make successful surgical suturing a most un-

certain procedure. Thus, while digital dilation has been undertaken for aortic stenosis,[31] we do not recommend it. We much prefer that one of our special curved aortic valve dilators be inserted through the tip of the left ventricle and passed up along the septum into the aortic valve (Figure 13). Care in passage, and external palpation of the root of the aorta with the left hand, will permit easy, safe dilatation of the stenosis. The valve will practically always fracture along one or both of the posterolateral commissures, this producing increased valve function by dilatation commissurotomy.[36]

6) *Correction of Auricular Septal Defects.*

The tremendous left to right shunt through auricular septal defects produces great overwork of the heart leading to death at a relatively early age (40 years) from congestive heart failure. Swan, Maresh, Johnson and Warner[37] have devised and applied in human subjects a method of correction of auricular septal defects by invaginating the auricular appendages against the septum in such a way as to occlude the abnormal opening. They use polythene buttons over each appendage tip depending upon pressure of the buttons, maintained by tightly tied sutures, to produce tissue irritation and healing. He passes the suture from the tip of one appendage through the heart, through the septal defect, and out the other appendage, using a probe. The septal defect is located by probe palpation. We have used Swan's principle in two cases, but have modified his technique. Thus, using the left lateral approach, we first insert the right index finger through an incision in the tip of the left auricular appendage and locate the auricular septal defect (Figure 14). The curved flat probe is then passed along the finger and is guided through the defect. Its tip is maneuvered up into and finally through the apex of the right auricular appendage. The suture is grasped and the probe is withdrawn. The finger is withdrawn from the heart, and the incision in the left appendage is sutured. The transcardiac sutures are then threaded on buttons of bone or cartilage prepared from the exposed rib cage. The invagination of the appendages is carried out exactly as Swan does (Figure 15).

7) *Ventricular Septal Defects.*

Ventricular septal defects of either small or of moderate size, high or low, may also be corrected by closed intracardiac tactile surgery. Large defects may, one day be corrected by a modification of this technique, but we have yet to undertake such a procedure in a human case. High ventricular septal defects not only permit a considerable left to right shunt with its attendant cardiac over-

work, but also permit a specific pulmonary vascular hypertension and pulmonary valve dilatation. This eventually leads to progressive pulmonary parenchymal and vascular changes which eventually interfere with oxygen absorption. Eisenmenger's syndrome may well have such an etiological mechanism (Figure 16).

Low defects in the muscular septum are less serious, especially since the muscular septum constricts the defect during systole, thus lessening the left to right shunt which can only take place during systole (Figure 17). However, they undoubtedly, like other intracardiac and even pulmonary and peripheral arterio-venous shunts, greatly diminish endocardial resistance to both bacterial and rheumatic infection. Thus, both Abbott[38] and Taussig[39] have shown an extremely high incidence of associated rheumatic infection in their respective series of cardiac septal defects. Lillihei[40] is of the opinion that this loss of resistance bears a straight line relationship to the size of the shunt.

Having decided to correct a ventricular septal defect, the heart is exposed by an anterior approach in the third left interspace. The pericardium is opened just anterior to the left phrenic nerve, and the anterior surface of the right ventricle is palpated to determine the location of the thrill—the site of the systolic jet of blood striking against the internal surface of the ventricular wall. An incision is made directly over this area after placing a purse string suture about the site. Either the left little finger or the left index finger is promptly inserted into the right ventricle and the defect is located by palpating the systolic jet of blood escaping from the left ventricle. Its size is estimated, and the finger is removed from the heart. Temporary hemostasis is afforded by tightening the purse string. A pedicled pericardial graft is then prepared in a manner similar to that used for mitral regurgitation. An attempt is made to make the graft large enough in its midportion to completely block the septal defect. Since no laxity is necessary, the flap may be extended merely as far as the diaphragm, rather than spiralling the heart as for mitral regurgitation (Figure 18a). The free end of the graft may be quite tapering, and is attached by a long heavy silk suture to a sharply curved probe. The finger is reinserted into the right ventricle, and the probe is passed along it into the ventricle and is guided through the septal defect. The tip of the probe is now in the left ventricle. It is redirected so that it runs diagonally along the left side of the ventricular septum, gradually approaching the anterior wall of the left ventricle. When it emerges from the anterior wall of the left ventricle, the suture is grasped and cut away from the probe (Figure 18b). The probe is withdrawn from the heart, and the sutures are drawn upon. As the tip of the graft is drawn into the right ventricle,

the intracardiac finger is withdrawn and the graft is pulled rapidly through the septal defect, and out through the anterior wall of the left ventricle (Figures 19a and 19b). If the graft is of suitable size, it tightly occludes the septal defect, high or low, like a cork in a bottle. Within a few hours, additional swelling occurs, producing further irritation to stimulate tight adherence and permanent healing (Figure 20).

Such a graft even in high defects does not lie in nor appreciably diminish the right ventricular outflow tract. It does not distort the aortic valve leaflets which make up the upper edge of the defect. It only slightly diminishes the left ventricular outflow tract. While we have only operated upon one high ventricular septal defect in a human case, to date, the results were so satisfactory in that one and the technique both in human and animals is so simple and logical, that we have no hesitance in recommending its wide application.

8) *Intracardiac Tumors.*

Intracardiac tumors of a benign type may occur in the auricular chambers, attached to the septal wall. We have operated upon one such case in which a tumor arising from the septum grew inside the left auricle until it partially blocked the mitral orifice, producing all the classical signs of mitral stenosis. It was possible to introduce a finger through the left auricular appendage so as to digitally dissect the tumor free from the auricular septum, and to express it away through the opening in the auricular appendage. Unfortunately, an inadvertant laceration of the auricular wall resulted in severe hemorrhage and death. It is the considered opinion of the authors that a slight modification of the employed techniques would render this procedure relatively safe and highly effective (Figures 21a and 21b).

These eight different types of procedure have already been performed in living humans, using the closed intracardiac tactile technique. In all, digital or tactile "vision" has proved satisfactory. Unquestionably, further developments along this line are imminent. Surely, some of the septal defects of Tetralogy of Fallot and of Eisenmenger's syndrome will be corrected, perhaps in the former instance at the same time as the direct correction of the pulmonary stenosis. Surely Lutenbacher's syndrome (mitral stenosis plus an auricular septal defect) will be subjected to simultaneous commissurotomy and septalcorrection. Intracardiac vegetations may eventually be removed by some such technique.

SUMMARY

Closed intracardiac surgery guided by the finger is a safe, logical, and highly efficient method of diagnosing and correcting most

of the common, acquired and congenital cardiac defects. Its applicability will undoubtedly shortly be extended to include other less common lesions. Its accuracy depends upon digital "vision" and digitally guided manipulations within the beating, functioning heart. The permissible maneuvers are obviously limited to certain relatively simple ones. Fortunately, most of the common intracardiac lesions can be corrected by the maneuvers now available. Care must be taken that the cardiac function is not seriously nor long impaired by the instrumentation.

While the eventual perfection of a suitable "heart-lung" machine may open many new possibilities in heart surgery, even then, the logic, the simplicity and the inherent safety of closed intracardiac tactile surgery, will certainly make it the surgeon's choice in the vast majority of intracardiac operations.

RESUMEN

Lacirugía cardiaca, guiada por el dedo, es segura, lógica y muy eficiente como método para diagnosticar y corregir la mayoría de los defectos comunes del corazón, adquiridos o congénitos. Su campo de aplicación, sin duda que se ensanchará pronto para incluir otras lesiones menos comunes. Su exactitud depende de la "visión" digital y de las manipulaciones seguidas dentro del corazón funcionando y palpitante. Las maniobras permisibles están obviamente limitadas a ciertas de ellas, relativamente sencillas.

Afortunadamente la mayoría de las lesiones intracardiacas pueden corregirse por las maniobras ahora posibles.

Debe tenerse cuidado de que la función cardiaca no sea seriamente interferida ni por largo tiempo, por los instrumentos.

Si bien, el eventual perfeccionamiento de una máquina "corazón-pulmón" puede abrir muchas posibilidades a la cirugía del corazón, aun entonces la lógica y sencillez, asi como la inherente seguridad de la cirugía cerrada, tactil o intracardiaca, hará que el cirujano la escoja en la mayoría de las operaciones intracardiacas.

RESUME

La chirurgie intra-cardiaque en se guidant sur le doigt est une méthode sûre, logique, et hautement efficace. Elle permet le diagnostic et le traitement de la plupart des anomalies cardiaques acquises ou congénitales, que l'on peut observer habituellement. Elle sera certainement dans peu de temps utilisée pour le traitement des lésions moins communes. La précision de cette technique est fournie par la "vision digitale" et les manipulations et en pleine fauction sous le contrôle du doigt à l'intérieur du coeur battant et en pleine fonction. Il n'en reste pas moins que les

manoeuvres que l'on peut se permettre sont limitées à certains cas relativement simples. Fort heureusement, la plupart des lésions intra-cardiaques habituelles peuvent être traitées par les manoeuvres que l'on peut actuellement utiliser. Il faut prendre soin que la fonction cardiaque ne risque pas d'être lésée par des pratiques traumatisantes ou trop prolongées.

On peut envisager que la perfection d'un couple "coeur-poumon" artificiel puisse un jour ou l'autre ouvrir de nouvelles possibilités dans la chirurgie cardiaque, mais, même en attendant, la simplicité et la sûreté de la chirurgie tactile intra-cardiaque en ferant certainement l'opération de choix dans la plupart des cas.

REFERENCES

1 Rehn, L.: "Suturing of Penetrating Wounds of the Heart," *Annals of Surg.*, 28:669, 1898.
2 Delorme: "Sur un traitement chirurgical, de la symphuse cardopeticardique," *Bull. et mem. Soc. de chit. de Par.*, 1898, n.s. xxiv, 918.
3 Brauer, L.: "Die Kardiolysis und ihre indikationen," *Arch. f. Klinic, Chir.*, 71:258, 1923.
4 Graybiel, A., Streider, J. W. and Boyer, N. H.: "An Attempt to Obliterate the Patent Ductus Arteriosus in a Patient with Subacute Bacterial Endocarditis," *Am. Heart J.*, 15:621, 1938.
5 Gross, R. E.: "Experiences with Surgical Treatment in 10 Cases of Patent Ductus Arteriosus," *J.A.M.A.*, 115:1257, 1940.
6 Crafoord, C. and Nylin, G.: "Congenital Coarctation of the Aortic and Its Surgical Treatment," *J. Thor. Surg.*, 14:347, 1945.
7 Gross, R. E.: "Surgical Treatment for Abnormalities of the Heart and Great Vessels," ed. 2, Springfield, Ill., Chas. C. Thomas, Pub., 1950.
8 Blalock, A. and Taussig, H. B.: "Surgical Treatment of Malformations of Heart in Which there is Pulmonary Stenosis or Pulmonary Atresia," *Journal and etc., etc.*
9 Potts, W. J., Smith, S. and Gibson, S.: "Anastomoses of the Aorta to a Pulmonary Artery for Certain Types of Congenital Heart Disease," *J.A.M.A.*, 132:627, 1946.
10 Doyen, E.: "Chirugie des Malformation Congenitales ou acquises du coeur," *Presse Med.*, 21:860, 1913.
11 Cutler, E. C. and Levine, A. S.: "Cardiotomy and Valvulotomy for Mitral Stenosis," *Boston M. and S. J.*, 188:1023, 1923.
12 Pribram, B. O. A.: "Die Operative behandlung der mitralstenose," *Auch. F. Clin. Chur.*, 142:458, 1925.
13 Allen, D. S. and Graham, E. A.: "Intracardiac Surgery: A New Method," *J.A.M.A.*, 79:1028, 1922.
14 Cutler, E. C., Levine, A. S. and Beck, C.: "The Surgical Treatment of Mitral Stenosis: Experimental and Clinical Studies," *Arch. Surg.*, 9: 689, 1924.
15 Souttar, H. S.: "The Surgical Treatment of Mitral Stenosis," *Brit. M. J.*, 2:603, 1925.
16 Bailey, C. P.: "Mitral Commissurotomy," *Dis. of Chest*, 15:377, 1949.
17 Smithy, H. G.: "An Approach to the Surgical Treatment of Chronic Valvular Diseases of the Heart," XVI, Annual Assembly of the Southeastern Surgical Conference, Hollywood, Fla., April 5-8, 1948.
18 Harken, D. E., Ellis, L. B., Ware, P. F. and Norman, L. R.: "The Surgical Treatment of Mitral Stenosis," *New England J. Med.*, 239:801 ,1948.
19 Murray, Gordon: "Treatment of Mitral Valve Stenosis by Resection and Replacement of Valve Under Direct Vision," *Arch. Surg.*, 61:903, 1950.
20 Sweet, R. H. and Bland, E. F.: "Surgical Relief of Concestion in the Pulmonary Circulation in Cases of Severe Mitral Stenosis," *Annals Surg.*, 130:384, 1949.
21 Brock, R. C.: "Pulmonary Valvulotomy for Relief of Congenital Pulmonary Stenosis," *Brit. Med. Jour.*, 1:1121, 1948.

22 Sellors, T. H.: "Surgery of Pulmonic Stenosis," *Lancet*, 988-989, June 26, 1948.
23 Blalock, A. and Kieffer, R. F. Jr.: "Valvulotomy for Relief of Congenital Valvular Pulmonic Stenosis," *Jour. Am. Surg. Ass'n.*, Vol. 68, 1950.
24 Downing, D. F., Bailey, C. P. and Glover, R. P.: "Brock Procedure for the Relief of Pulmonary Stenosis in Tetralogy of Fallot," *Pediatrics*, Vol. 7, No. 2, Feb. 1951.
25 Glover, R. P., Bailey, C. P., O'Neill, T. J., Downing, D. and Wells, C. R. E.: "The Direct Intracardiac Relief of Pulmonary Stenosis in Tetralogy of Fallot," 31st Annual Meeting, Amer. Ass'n. for Thor. Surg., April 16-18, 1951. To be published.
26 Deaver, John: Personal communication.
27 Murray, G.: Discussion of "Mitral Commissurotomy," *Dis. of Chest*, 15:377, 1949.
28 Bloomberg, Allen: Discussion of "Experiences in Surgery of Aortic Stenosis," *J. Thor. Surg.*, 20:516, 1950.
29 Bailey, C. P., O'Neill, T. J., Glover, R. P., Jamison, W. L. and Ramirez, Hector R. R.: "Surgical Repair of Mitral Insufficiency (A Preliminary Report)," *Dis. of Chest*, 19:125, 1951.
30 Karlson, K. E., Dennis, C., Westover, D. and Sanderson, D.: "Pump-oxygenator to Supplant the Heart and Lungs for Brief Periods," *Surg.*, 29:678, 1951.
31 Harken, D. E.: Personal communication.
32 Bailey, C. P., Glover, R. P. and O'Neill, T. J. E.: "The Surgery of Mitral Stenosis," *J. Thor. Surg.*, 19:16, 1950.
33 O'Neill, T. J. E., Glover, R. P. and Bailey, C. P.: "Observations on the Surgical Treatment of Mitral Stenosis by Commissurotomy," *J.A.M.A.*, To be published.
34 Bailey, Charles P., Lacy, M. M. and Harris, J. S. C.: "The Surgical Treatment of Acquired Heart Disease," *Surg. Clin. N. A.*, Vol. 31, No. 6, December 1951.
35 Satinsky, Victor: Personal communication.
36 Bailey, C. P., Glover, R. P., O'Neill, T. J. E. and Ramirez, H. P.: "Experiences with the Experimental Relief of Aortic Stenosis," *J. Thor. Surg.*, 20:516, 1950.
37 Swan, H., Maresh, George, Johnson, Marvin and Warner, George: "The Experimental Creation and Closure of Auricular Septal Defects," *J. Thor. Surg.*, 20:542, 1950.
38 Abbott, Maude: "Atlas of Congenital Heart Disease," N. Y. 1936, Amer. Heart Association.
39 Taussig, Helen: "Congenital Malformation of the Heart," N. Y. 1947, The Commonwealth Fund.
40 Lillihi, Clarence: Personal communication.

Physico-Chemical Pneumonitis*

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The lung may be seriously damaged by "nonbiotic" or physical and chemical insults. These agents usually cause a type of inflammation which should be called "pneumonitis" and not "pneumonia." Pneumonia[1] is a self-limited inflammation of the lung tending to heal without significant pulmonary damage; it is characterized mainly by intraalveolar exudate although there may be minor secondary interstitial changes. Pneumonitis means inflammation of the lung tending to be recurrent and chronic, with profound interstitial and intrabronchial involvement as well as intra-alveolar exudate often resulting in suppuration and fibrosis. Organization and fibrosis are the end results, with permanent pulmonary damage.

Several impressive varieties of physio-chemical pneumonitis have been reported, but basically these represent "nonspecific" responses of the lung to a variety of agents. Whether the patient has a mild syndrome resulting in little incapacity or severe pulmonary damage depends chiefly on the interplay of two factors; intensity of the insult and strength of the body defenses. An accurately taken history is needed to determine the etiology; a technically acceptable roentgenogram to estimate lung damage, and a meticulous physical examination, including physiologic estimations to measure pulmonary function. Only serial study suffices to follow the course of the disease.

Several diagnostic pitfalls must be avoided. The resemblance among these syndromes is so great that it is fallacious, on the basis of the roentgenograms alone, to attempt to differentiate lipoid pneumonitis from the pulmonary necrosis caused by aspirated gastric hydrochloric acid. Similarly, to distinguish chronic pneumonitis of any type from carcinoma or from tuberculosis is not always easy. Only by thorough clinical investigation is it possible to establish an adequate diagnosis.

Although these various forms of pneumonitis have much in common, several of them are sufficiently distinctive to justify separate mention. The physical and chemical factors capable of inflaming the lung are many but usually act through aspiration, inhalation, irradiation or trauma. Most forms of pneumonitis

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terminate with the common factor of fibrosis sufficient to cause prolonged illness or even death. In this respect, physio-chemical pneumonitis is a syndrome with many features similar to "chronic pneumonia."[2]

A. *Aspiration*

Aspiration pneumonitis in all its forms occurs only after something overwhelms the natural defenses of the tracheo-bronchial tree which normally prevent entrance of foreign material. If the aspirated material is retained long enough in the lower airways, acute exudative inflammation will occur. The pulmonary defenses are obviously weak in the comatose, narcotized or anesthetized patients less obviously impaired wherever cough reflex and bronchial drainage are not effective. Several examples of the effects of aspirating foreign materials into the lung illustrate the permanent pulmonary damage that may be produced by the combination of bronchial obstruction, chemical injury, and subsequent bacterial invasion with ultimate fibrosis.

One case was that of a four year old boy who aspirated vomitus during anesthesia for appendectomy. Prompt administration of antibiotics and oxygen was followed by complete resolution of the pneumonitis within four days' time. Presumably the gastric hydrochloric acid causes pulmonary damage which is reparable if not too extensive, and if infection is combatted vigorously. Figure 1 illustrates the x-ray film of a 46 year old woman whose car overturned in a swamp causing submersion for about two minutes. She was comatose and almost asphyxiated when seen 20 minutes after the accident. A film made within an hour after treatment was begun (with suction, nasally administered oxygen and antibiotics), shows a right basal pneumonitis associated with pulmon-

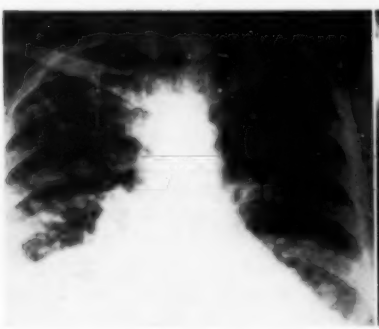

FIGURE 1

FIGURE 2

ary edema. A film made six weeks later showed residual fibrosis although she had few symptoms.

Ramagosa, Menville, and Leckert[3] reported the case of a man rescued from drowning in a salt water swimming pool. An x-ray film 36 hours after resuscitation showed mottled opacities with areas of confluence in the inner two-thirds of the lung fields and in both pericardial areas. The peripheral lung fields appeared clear. Subsequent progressive clearing was complete at the end of two weeks. These changes were attributed to pulmonary edema with atelectasis as an associated factor.

Aspiration of oil[4] may occur as a result of the use of oily nose drops, administering oily substances to the struggling patient or through regurgitation. Liquid petrolatum, milk fat, and cod liver oil are the most frequent offenders, causing a pneumonitis affecting chiefly the bases and posterior portions of the lungs, especially the right lung. Infiltrations may extend from the hilar areas into both lung fields simulating tuberculosis, but the apices are usually free. Particularly in children, virus pneumonia may be simulated by involvement of the inner and middle parts of the lungs, but the chronicity of the infiltrates is a definite point of differentation. Larger agglomerations ("paraffinomas") may be mistaken for neoplasm. At times, the sputum contains oily droplets which may be demonstrated by the special technic of Lossner et al.,[10] or which may clearly mark a cigarette paper. Whether "cholesterol Pneumonitis" represents a special variety of lipoid pneumonitis has not been determined.[6] Experimentally, it can be shown that this inflammation starts within a few minutes after oil has reached the lungs, but is seldom elicited by those oily substances used for bronchography. Figure 2 represents the film of a 45 year old man who had chronic undiagnosed right middle lobe lesion. Bronchoscopy and bronchography were not helpful; since abnormal cells were several times found in the sputum, a tentative diagnosis of bronchogenic carcinoma was made and pneumonectomy performed. In the excised lung, a "paraffinoma" was demonstrated. Subsequently, the history of prolonged use of "mineral oil" was obtained.

Heacock[5] reports a classical case of aspiration pneumonitis following ingestion of kerosene which, being of low volatility tends to remain in the lungs. Fatality was early; postmortem examination performed two hours after ingestion of kerosene showed generalized, confluent hemorrhagic edema. Another case is that of a 26 year old seaman who was blowing out a clogged gas line with his mouth while standing in gasoline a foot deep on a ship. He slipped, fell to the floor, and lay unconscious in the gasoline about 40 minutes before he was discovered and carried to the

hospital. He suffered extensive chemical burns, and became violently psychotic before death, 30 hours after the accident. At post-mortem examination, extensive bilateral focal hemorrhagic pulmonary edema was found. A 17 months old girl who, 31 hours before admission, had ingested kerosene, promptly regurgitated and aspirated some of it. She recovered after antibiotics had been administered, but residual pneumonitis was demonstrable two weeks later.

B. *Inhalation of Irritants*

The inhalation of an irritating gas causes immediate inflammation of the respiratory tract varying from superficial erythema of the mucosa of the upper air passages to a fatal pulmonary edema. Usually there is an outpouring of thin fluid which at times is copious enough to cause asphyxia. As a result of chemical irritation there is immediate stimulation of the lymphatic flow, clearing the foreign particles and edema fluid from the alveoli

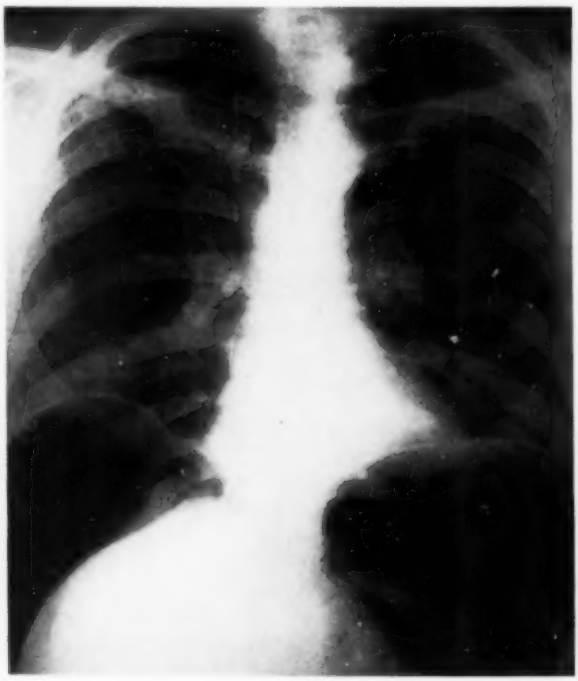

FIGURE 3

allowing oxygen-carbon dioxide transfer. If the resulting edema is severe enough to prevent gaseous transfer, anoxia results. The latter may be fleeting or may lead to death, depending on the intensity and duration of the reaction. Bronchial obstruction at first is caused by muscle spasm, but soon exudate, hemorrhage, and debris block the larger air passages. The diffusion of oxygen is further impeded by thickening of the alveolar membrane. If the individual does not drown in the edema fluid, the anoxia is compensated by an increase in the output of the heart; but if the heart has to carry an unbearable load from increasing resistance of the lesser circulation, cardiac failure results.

The lungs are usually large, boggy, and deeply congested and on section reveal areas of edema, coagulation necrosis, hemorrhage, thrombosis, and infarction. If death does not occur early, the lungs may reveal inflammatory changes and suppurative foci caused by secondary organisms. Milder degrees of irritation give rise to a condition of chronic pneumonitis.

The symptoms and physical findings of acute gas poisoning resemble those of an asthmatic paroxysm, a feature being the presence of large bubbling edema rales and crepitation. The roentgenogram reveals soft conglomerations chiefly in the lower and midparts of the lung much as in acute pulmonary congestion but the nodules are more distinct, and the cardiac silhouette is unaltered. Since the nodules represent pulmonary edema, serial examinations reveal rapid clearing if the patient survives. In the presence of secondary infection, the roentgen findings are the same as those encountered in other forms of bronchopneumonia.

The Cocoanut Grove disaster of Boston provided an opportunity

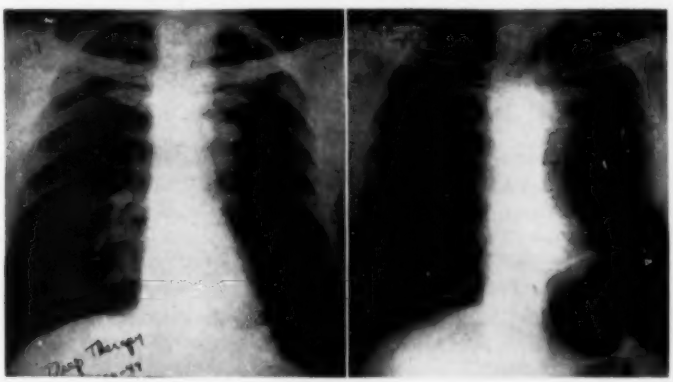

FIGURE 4

of study of this sort of respiratory damage caused by hot irritating fumes. The clinical manifestations ranged from laryngotracheal bronchitis to varying degrees of severe pneumonia. The latter was associated with bronchial obstruction due to a pseudomembranous exudate which resulted in a mixture of obstructive emphysema and atelectasis. The areas of atelectasis were represented by triangles or bands or disks involving chiefly the lower lobe, emphysema of a lobar or lobular distribution, miliary and larger areas of mottling caused by atelectasis, and fluid and pulmonary edema.

Beryllium fumes may cause an acute pneumonitis; recovery is the rule within a few weeks of discontinuing exposure with relapse on re-exposure. The chronic form of beryllium granulomatosis is unlike pneumoconiosis and is marked by dyspnea and cough, is associated with extensive emphysema and fibrosis, bears no signs of an infectious process and may be uninterrupted by remission. During life the diagnosis must usually be based on roentgen findings on a person exposed to beryllium. Beryllium can be found in excised postmortem tissues. The structural features of the granulomata tend to differentiate them from silicosis and from silicotuberculosis, while the inclusion bodies must be differentiated from sarcoid. The roentgen findings are miliary and discrete larger nodules with composite nodular densities and connective tissue infiltrate. Diffuse miliary nodulation is early and resembles the lesions of Q fever and of Friedlander's infection. Later there is a reticulo-nodular infiltrate. Deposits of calcium apparently represent the end phase of the evolution of the lesion. Multiple bleb formation was observed in all reported cases, twice leading to spontaneous pneumothorax. Cor pulmonale led to congestive heart failure in one case. Figure 3 shows the x-ray film of a 45 year old Negro janitor who had for many years broken fluorescent electric light tubes. The chronic pulmonary granulomatosis had produced a marked degree of dyspnea for which pneumoperitoneum was administered with moderate relief.

C. *Irradiation*

Irradiation of the thorax, especially for malignant disease of the esophagus or of the breast, may cause chronic pneumonitis at times confused with the original disease for which irradiation was conducted. Experimentally, both immediate hyperemia and delayed onset of fibrosis may be produced by irradiating animals. In humans, the symptoms of pulmonary disease may appear while treatment is in progress. Pleural fibrosis is minimal, but parenchymal fibrosis is extensive and may lead to mediastinal retraction with distortion. The earliest visualized changes are increased radiolucency of the chest followed after a few weeks by formation

of fibrous bands with retraction phenomena. In metastatic malignancy, the lesions usually do not resolve, and are often accompanied by pleural effusion; shrinking of the affected hemithorax seldom occurs. A 49 year old woman with lymphoblastoma had lesions that resolved following irradiation of the chest. Pulmonary fibrosis subsequently developed and was accompanied by the usual retraction phenomena. Here the original malignancy partially obscures the irradiation pneumonitis. Figure 4 shows the x-ray film of a 50 year old man with an undiagnosed mediastinal mass for which surgery was rejected. Following irradiation, the mass diminished in size, but nine months later there was well established fibrosis of the left upper lobe.

D. *Trauma*

There have already been several case reports of injury to the lung (particularly "blast injury") among persons who did not sustain rib fractures or penetrating chest wounds. The mechanism of injury predisposes to pulmonary edema and hemorrhagic necrosis; the victim survives the initial onslaught, he is apt subsequently to develop pulmonary fibrosis through repair of the damaged lung.

SUMMARY

The lung may be damaged by an ever-increasing number of physical and chemical forms of violence. This violence may come from the hazards of industry, from attempts to treat other disease, or from the environmental dangers of current existence. Regardless of the etiology, the syndrome of pneumonitis with its sequellae of suppuration and fibrosis may occur. Once such a syndrome has been produced, it should be treated energetically by removal of the offending agent if possible, by combatting bacterial infection, by stimulating body defenses, and by enhancing the self-cleansing action of the tracheobronchial tree.

RESUMEN

El pulmón puede ser dañado por un número cada día mayor de formas de traumas, físicos y químicos. Tal lesión puede provenir de los riesgos de la industria, de los esfuerzos por curar otras enfermedades, o de los peligros ambientales de la vida actual. Sea cual fuere su etiología, puede sobrevenir el síndrome de la neumonitis, con su secuela de supuración y fibrosis. Una vez que se haya producido tal síndrome, debe tratarse enérgicamente, eliminando el agente dañino, si fuera posible, combatiendo la infección bacteriana, estimulando las defensas orgánicas, y aumentando la función espontánea de eliminación, del árbol traqueo-bronquial.

RESUME

Le poumon peut être atteint par des agressions de substances physiques ou chimiques en nombre toujours croissant. Ces agressions peuvent provenir des conditions du travail industriel, des agents thérapeutiques utilisés contre d'autres affections, ou des dangers habituels de l'existence. Quelle que soit l'étiologie on peut voir apparaitre le syndrome d'atteinte pulmonaire, et ses séquelles de suppuration et de sclérose. Quand un tel syndrome est constitué, il faut le traiter avec énergie. Pour ce traitement, il faut supprimer si possible l'agent déterminant, il faut combattre l'infection bactérienne, il faut stimuler la défense de l'organisme et il faut favoriser l'action d'épuration spontanée qui appartient à l'arbre trachéo-bronchique.

REFERENCES

1 Pinner, M.: "Pneumonitis," *Am. Rev. Tuberc.*, 38:730, 1938.
2 Jacobs, S.: "Chronic Pneumonias," *New Orleans M. and S. J.*, 100:207, 1947.
3 Romagosa, J., Menvile, L. J. and Leckert, J. T.: "Radiographic Changes in Lungs During Recovery from Drowning," *Radiology*, 55:517, 1950.
4 Freiman, D. G., Engelberg, H. and Merrit, W. H.: "Oil Aspiration (Lipoid) Pneumonia in Adults," *Arch. Int. Med.*, 66:11, 1940.
5 Heacock, C. H.: "Pneumonia in Children Following the Ingestion of Petroleum Products," *Radiology*, 53:793, 1949.
6 Waddell, R., Sniffen, R. C. and Sweet, R. H.: "Chronic Pneumonitis; Its Clinical and Pathological Importance. Report of Ten Cases Showing Interstitial Pneumonitis and Unusual Cholesterol Deposits," *J. Thoracic Surgery*, 18:707, 1949.
7 Reiman, H.: "The Pneumonias," Philadelphia, *W. B. Saunders Company*, 1938.
8 Robert, A. G.: "A Consideration of the Roentgen Diagnosis of Chronic Pulmonary Granulomatosis of Beryllium Workers," *Am. J. Roent.*, 63:467, 1950.
9 Warren, S. and Spencer, J.: "Radiation Reaction in the Lung," *Am. J. Roent.*, 43:682, 1940.
10 Lossner, S., Volk, B. W., Slade, W. R., Nathanson, L. and Jacobi, M.: "Diagnosis of Lipoid Pneumonia by Examination of Sputum," *Am. J. Clin. Path.*, 20:539, 1950.

Recent Trends in Esophageal Surgery*

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In the past decade surgery of the esophagus has made tremendous strides, not only in the field of carcinoma but also in the numerous other conditions to which this organ is heir. Diseases of the esophagus fall not only into the realm of the surgeon but into that of the general practitioner, the internist, the pediatrician, the anesthetist, the roentgenologist and the otorhinolaryngolist as well. It is for this reason that a presentation dealing with this subject is timely.

Anatomy

Some points in the surgical anatomy of the esophagus bear emphasis. It is well to remember that the esophagus per *se* is 10 inches (25 cm.) in length, extending from the sixth cervical vertebra (cricoid cartilage level) to the 10th dorsal vertebra (esophageal hiatus level). The esophagologist, however, does not measure from the sixth cervical vertebra but rather from the alveolar margin, which adds an additional six inches (15 cm.) to its actual length; thus he bases his report upon a 16-inch (40 cm.) structure.

The esophagus has three normal curves and three physiologic constrictions. Its three curves or flexures are placed in the following manner: in the neck it curves to the left, as it descends in the thorax it curves to the right, and as it approaches the esophageal hiatus it curves again to the left. These flexures are of practical importance, since they may determine the proper side of approach. The three physiologic constrictions are located (1) at the level of the cricoid cartilage (sixth cervical level), (2) at the arch of the aorta and (3) at the esophageal hiatus. It is at these constrictions that the greatest amount of irritation takes place and the greater number of pathologic lesions are observed. Most foreign bodies are impeded in their path downward at the first physiologic constriction and are therefore found on a level with the sixth cervical vertebra. In locating a swallowed foreign body it is all-important, therefore, to order not only a roentgenogram of the chest but one of the neck as well.

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From the Departments of Surgery, University of Illinois, Cook County Graduate School of Medicine, American and Alexian Brothers' Hospital.

The relation of the vagus nerves to the esophagus should be stressed, since these nerves may be involved or must be sacrificed in some esophageal procedures. Although much has been written recently regarding the anatomy of the vagi and although numerous patterns have been described, in the main it may be stated that the left vagus nerve passes along the anterior aspect of the esophagus, intimately hugs the food pipe and is the smaller of the two vagi. The right vagus passes posteriorly, does not hug the esophagus and is larger.

To know the boundaries of the so-called "esophageal triangle" is to make identification and isolation of the supradiaphragmatic portion of the esophagus a simple and rapid maneuver. This anatomic triangle is bounded in front by the heart, behind by the descending aorta and below by the diaphragm.

Almost every text, monograph or recent article calls attention to the poor blood supply of the esophagus. I cannot agree with this teaching. To emphasize this point one needs only to divide

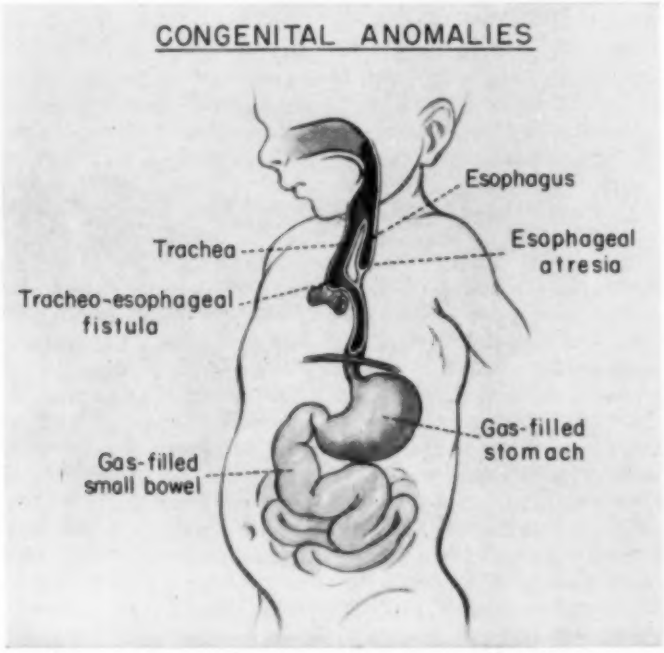

FIGURE 1: The esophageal atresia involves the proximal segment of esophagus and the tracheo-esophageal fistula involves the distal end of the esophagus. The gas in the stomach and small bowel is derived from the trachea.

the esophagus into upper (cervical), middle (thoracic) and lower (abdominal) parts. The upper part of the organ is supplied voluminously by the inferior thyroid artery; the middle or thoracic part is supplied by aortic, bronchial and intercostal branches; the lower part is well supplied by the left gastric and the inferior phrenic arteries. In my work I have demonstrated, and also presented in a recent motion picture of the actual procedure, the removal of the entire thoracic esophagus, both supra-aortic and infra-aortic portions, from its mediastinal bed. After this the esophagus was transected at its entrance into the stomach, and brisk, bright red arterial oozing was noted. I feel, therefore, that much more can be expected of the esophagus in its ability to heal, particularly if careful sutures are placed and if tension is avoided.

Atresias and Tracheo-Esophageal Fistulas

Various combinations and degrees of these anomalies are possible. The most common such defect is one in which the proximal end of the esophagus terminates as a blind pouch and the distal end joins the trachea to form a tracheo-esophageal fistula (Fig. 1).

Such a defect is to be suspected if the newborn baby presents a symptom complex revealing the three "C's," namely, *coughing, choking* and *cyanosis*. Especially is this true if there is an abnormal amount of drooling and rhinorrhea. These symptoms should suggest an impending surgical emergency. The roentgenologist is of inestimable value in determining the type and site of such lesions by merely noting the presence or absence of gas in the stomach and bowel, and by injecting a small amount of lipiodol through a nasal catheter into the proximal portion of the esophagus (barium should never be used).

The side of approach to atretic lesions and tracheo-esophageal fistulas is open to controversy. It is true that if only a small narrow atretic portion in the esophagus exists the right-sided approach is preferable. The reasons for this are that the arch of the aorta does not interfere with exposure, only the azygos vein need be ligated and severed to visualize the esophagus thoroughly, and an end-to-end anastomosis may be accomplished readily. However, if the atresia or the fistula involves a longer segment of esophagus, an end-to-end esophageal anastomosis cannot be accomplished. Thus if one has utilized a left-sided approach it is an easy matter to open the diaphragm, mobilize the stomach, and perform an intrathoracic esophagogastrostomy. It should be remembered also that in the newborn and in the young the aorta is not the barricading impediment that it is in older persons, since in the young it can readily be retracted out of the way.

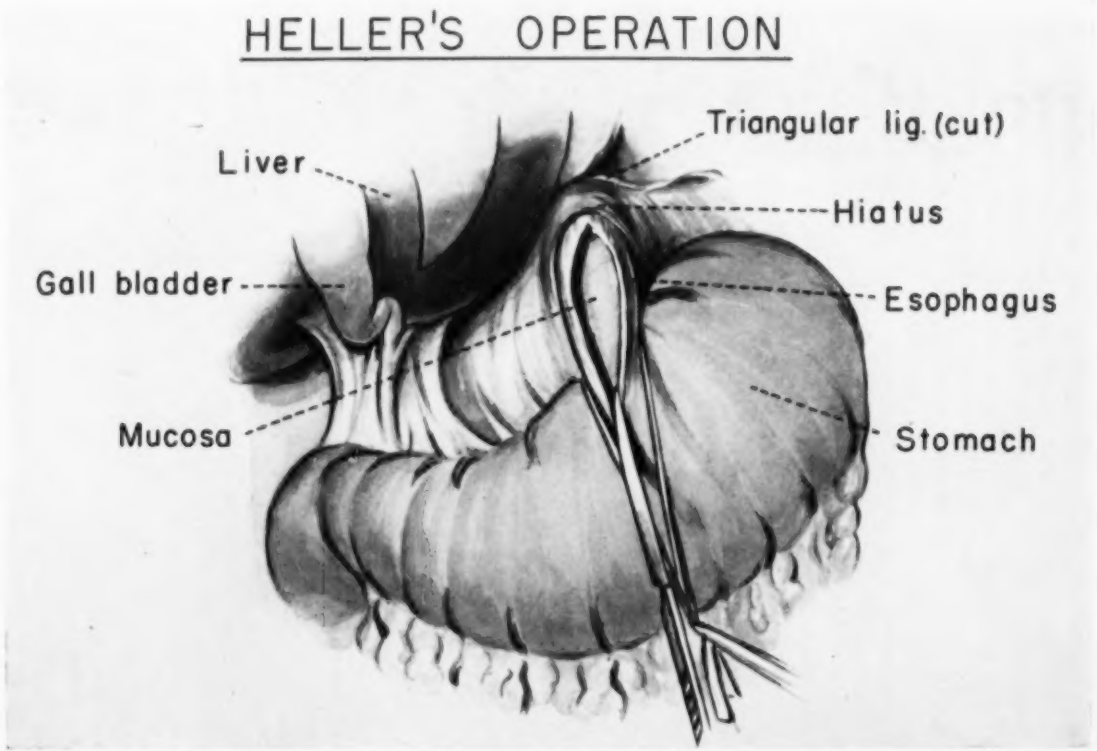

FIGURE 2: The left lobe of the liver has been reflected to the left after cutting the left triangular ligament. An extramucous esophagocardiomyotomy is performed.

If a few of these pertinent facts are kept in mind, not every "blue baby" will be thought to present the tetralogy of Fallot, and an immediate and proper work-up will be instituted, thus saving many such children from unnecessary deaths.

Diverticula of the Esophagus

Esophageal diverticula are classified into pulsion and traction types. Surgically we are interested in the pulsion type of diverticulum, which occurs either in the neck (cervical) or immediately above the diaphragm (epiphrenic). The so-called traction type is usually located in the region of the tracheal bifurcation where the chronically inflamed tracheal lymph nodes are found. These rarely produce symptoms, since they usually have a wide ostium which forms their neck. However, more important than the classification is appreciation of the pathologic physiology which underlies the formation of this condition. It is believed that one of the prerequisites for the pulsion type is a weakness in the muscular wall, which permits herniation of the mucosa through the weak part and results in a diverticulum. No symptoms are produced as long as the opening into the diverticulum is large. However, as food slowly packs into the diverticulum the latter increases in size and drops downward. This process continues until the weighted sac hangs downward and produces pressure upon the esophagus, obstructing it. It should be noted that in such far-advanced stage food has easier access to the diverticulum than to the esophageal lumen proper. Dysphagia is the outstanding complaint, and diverticulitis, perforation and mediastinitis are the more common complications.

In this day of chemotherapy, improved surgical technic, modern anesthesia and proper postoperative care, the one-stage diverticulectomy is preferred over the two-stage procedure. Although removal of the diverticulum is preferred, diverticulopexy still has its place. The latter procedure, in which the diverticulum is dissected free and stitched upward to the surrounding structures, is deserved for the poor risk patient with marked symptoms. When this has been done, food does not have access to the downward-hanging diverticulum and thus the symptoms are relieved.

Cardiospasm (Achalasia) of the Esophagus

Cardiospasm is neither cardiac nor spastic. It is a condition in which there is dilatation, hypertrophy and tortuosity of the esophagus unassociated with an obstructed esophageal hiatus. Theories as to its production are numerous and will not be discussed here. This condition is twice as common in females as in males, and in

a rather large percentage of cases a history of emotional strain or psychic trauma can be elicited.

The outstanding symptoms are dysphagia, regurgitation and pain. Complete esophageal obstruction for varying periods has been noted, even to a point where the patient cannot swallow his own saliva. The pain is usually retrosternal and varies from mild to severe. Morphine addiction has resulted from this condition.

The roentgen film is of particular help in diagnosing cardiospasm. Recently Kramer and Ingelfinger described the mecholyl test as an aid to the diagnosis of cardiospasm. When mecholyl, a parasympathomimetic drug, is injected into a normal person there is only a slight increase in tone and magnitude of esophageal contractions. However, in patients with cardiospasm, marked esophageal spasm and contractions follow administration of the drug. Complications that may be associated with the condition are bronchitis, bronchiectasis and pulmonary abscess. All of these may result from aspirated material regurgitated from the esophagus.

Medical management includes psychotherapy, antispasmodics and dilatation. Surgically many types of procedures have been advised, which include cardioplasties, cardiomyotomies, resections and anastomoses. The procedure of choice is Heller's operation, which is a transabdominal extramucous esophagocardiomyotomy. This is simple to do with a negligible morbidity and mortality. The postoperative results are excellent (Fig. 2).

Hiatus Hernia

The following three types of esophageal hiatal hernias have been described: (1) the para-esophageal type, in which the stomach herniates through the esophageal hiatus and lies parallel with an esophagus of normal length; (2) the sliding type, in which the stomach is herniated through the hiatus but has pushed an esophagus or normal length upward, thus rendering it tortuous, and (3) the hiatal hernia with a congenitally short esophagus, in which the upper part of the stomach is herniated through the hiatus because the esophagus has never attained its normal length.

The last-mentioned type can be diagnosed by the esophagologist when he records the appearance of gastric mucosa at an unusually high level, indicating a short esophagus. Hiatal hernias may be observed as a coincidental finding in taking gastro-intestinal roentgen films. Such hernias rarely if ever produce symptoms. Although many advocate leaving them alone, the danger of strangulation of such a herniated segment of stomach, with resulting putrid empyema and rapid death, must be kept in mind.

Many patients with hiatal hernias complain of epigastric distress, bloating, belching, anorexia, loss of weight and retrosternal pain. Such cases have been diagnosed erroneously as disease of the gallbladder, peptic ulcer, gastritis or coronary disease. These errors can be avoided if the possibility of hiatal hernia is kept in mind and if roentgen films are taken with the patient in the Trendelenburg position, which would readily demonstrate herniation that might otherwise be overlooked.

The surgical repair of a para-esophageal hernia or a sliding type of hernia is simple, since all that is necessary is reduction of the herniated stomach and tightening of the esophageal hiatus. It is the hernia associated with a congenitally short esophagus that presents a problem. For such a lesion I have devised a procedure by which the hiatus is incised and enlarged and the defect increased. This is accomplished by herniating more stomach into the thoracic cavity, thus removing the pull on the congenitally foreshortened esophagus. The esophageal hiatus is then loosely sutured around the stomach. In the poor risk patient phrenicotomy might produce gratifying results.

Portal Hypertension

This condition results from increased pressure in the portal system and is almost always observed at the lower end of the esophagus (recent studies reveal, however, that varices may occur in any part of the esophagus). Portal hypertension may be due to intrahepatic or extrahepatic causes. The usual condition causing intrahepatic block is cirrhosis of the liver. Extrahepatic block may be due to phlebitis, thrombosis, fibrous stenosis or cavernomatous transformation of the portal vein; it may be congenital or acquired.

One does not refer to Banti's disease in these modern times, but rather to Banti's syndrome. This is characterized by splenomegaly, anemia, esophageal varices, ascites and leukopenia. The syndrome has been produced experimentally by Whipple, who blocked the portal vein. If the block is due to an intrahepatic lesion the liver function tests will give positive results, whereas if the portal block is extrahepatic the results of these tests are usually negative. If the condition is in a quiescent stage it may be safe to take an esophagram, which will reveal the pathognomonic "beaded" appearance at the lower end of the esophagus.

Since hemorrhage from a ruptured esophageal varix may be fatal, definite measures must be taken to prevent recurrence. Some of the therapeutic procedures advocated are injection of the varix with sclerosing agents, tamponade with oxycel or gelfoam through the esophagoscope, ligation of the coronary vein,

esophagogastric resection to remove the "pile-bearing" area, and various forms of portacaval shunts. More recently splenic artery ligation, splenectomy, and hepatic artery ligation have been advocated. The author has performed three of the latter procedures following the suggestion of Rienhoff.

Portacaval shunts have attained a certain degree of popularity in the past few years, and various types of anastomoses have been attempted in order to shunt the blood from the portal to the caval system. It is my opinion that, rather than the time-honored end-to-side portacaval shunt (Eck fistula), a lateral anastomosis between the portal vein and the inferior vena cava will prove to be the method of choice. This is accomplished best through a right thoracolaparotomy incision in which the costal arch is divided, making it possible to dislocate the right lobe of the liver into the right thoracic cavity. When this approach is used, the portal vein and the inferior vena cava practically approximate themselves, thus avoiding the necessity of dissecting out the common duct and the hepatic artery. It seems preferable also to permit some portal blood to continue through the liver rather than make a complete division of the portal vein. Only time will tell the efficacy of these measures.

Carcinoma of the Esophagus

What was considered a hopeless and inoperable condition only a decade ago presents at least a worthwhile challenge to the doctor of today. If present impressions are correct, the overall outlook and prognosis for the patient with carcinoma of the esophagus is better than the 5 per cent, five-year "cure" for carcinoma of the stomach.

It is of practical value to divide the esophagus into zones. Many such divisions have been described. One that has served me well divides the organ into three zones, namely, an upper (Zone 1), which extends from the beginning of the cervical esophagus to the manubrium sterni; a middle (Zone 2), which extends from the manubrium sterni to the inferior pulmonary ligament, and a lower (Zone 3), from the inferior pulmonary ligament to the esophagogastric junction.

Early diagnosis is of paramount importance in cases of this type of carcinoma, since it is by this means that the survival rate can be increased. One ofter hears mention of a "change in stool habit" as a probable symptom of carcinoma in the lower intestinal tract; in like manner it may be stated that "any persistent change in the swallowing habit is an indication of carcinoma of the esophagus until proved otherwise." Were this dictum to be heeded and such cases further investigated by means of esophagoscopic and

roentgen study, then many esophageal neoplasms would be operated upon earlier.

Treatment

Needless to say, preoperative and postoperative therapy for these patients is of the utmost importance, and much has already been written concerning these phases.

For lesions located in Zone 1 the Wookey procedure has been used, especially for a tumor situated in the cervical portion of the esophagus. Recently, however, lesions involving the superior mediastinal segment of the esophagus as well as the cervical part have been approached by a more radical procedure, which removes the thoracic portion of the esophagus and most of the cervical portion. This is accomplished through a thoracic phase of the operation and a cervical phase. The operation is completed by an intracervical end-to-side esophagogastrostomy. In placing the mobilized stomach in the neck the surgeon may find that the thoracic inlet impinges upon the stomach. If this is the case it may be necessary to remove the inner half of the clavicle and the first rib. Whether the mobilized stomach should be placed behind or in front of the arch of the aorta is a moot question and must be decided by the surgeon at the time of operation.

Lesions of Zone 2, or those which involve most of the thoracic portion of the esophagus, are removed by a rather standardized procedure in which a wide esophageal resection is accomplished, followed by an end-to-side supra-aortic esophagogastrostomy or esophagojejunostomy (Fig. 3). When the jejunum is used it may be necessary to do a Roux "Y" anastomosis or some modification thereof.

Lesions of Zone 3 include not only the lower end of the esophagus but the gastric cardia as well. I prefer a thoracolaparotomy incision in which a left rectus incision extends across the left costal arch, dividing it and then continuing into the eighth intercostal interspace. The diaphragm is divided to and through the esophageal hiatus, and the esophagus is mobilized from the aortic arch to its entrance into the stomach; the latter is then mobilized to the pylorus. The right gastric and gastro-epiploic arteries should be left intact. An esophagogastric resection is then accomplished and is followed by an end-to-side esophagogastrostomy. If necessary the spleen and part of the pancreas are removed.

In most of my esophageal operations water seal drainage is preferred, and a Levine intragastric tube is permitted to remain in place for the first three postoperative days. I cannot subscribe to the teaching that these tubes may be harmful. Routine portable

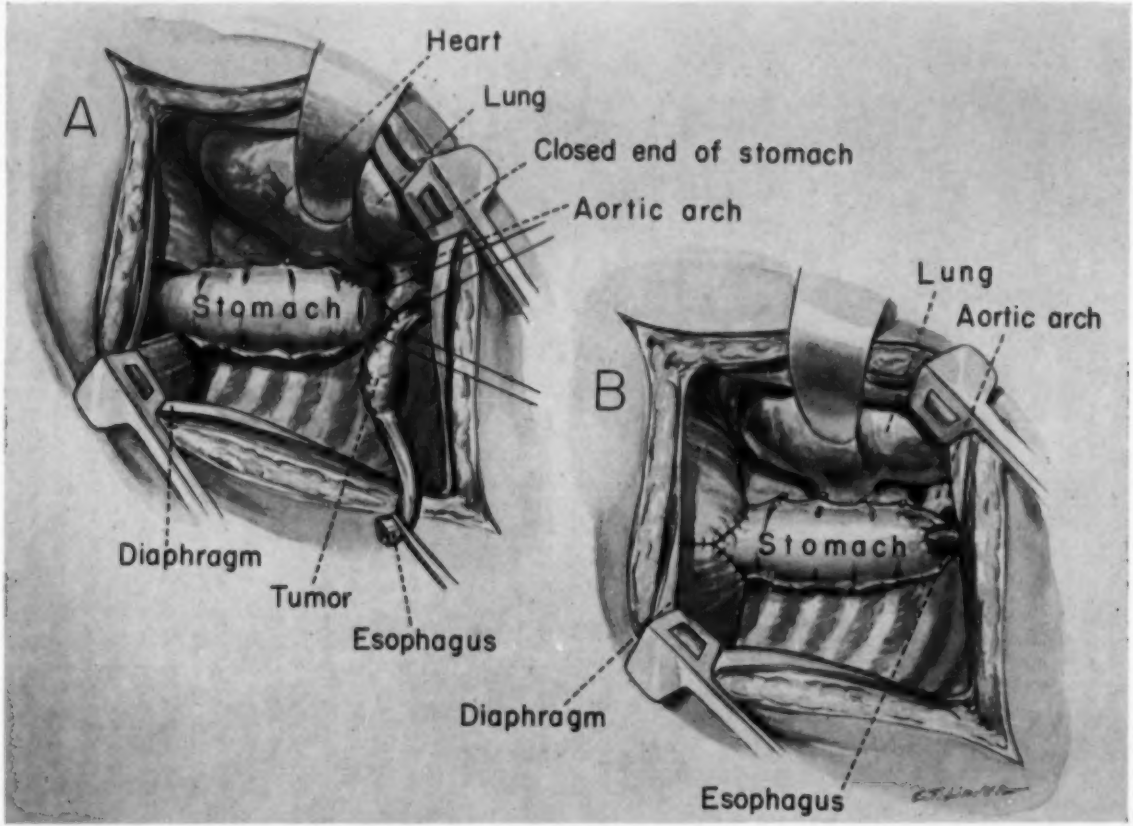

FIGURE 3: The Surgical Treatment for Zone 2 Carcinoma.—(A) The esophagus has been freed from the stomach and brought over the arch of the aorta.—(B) The esophagus and tumor have been resected and a supra-aortic end-to-side esophagogastrostomy completes the procedure.

roentgenograms are taken 24 hours after operation to determine the pulmonary status.

Palliative Procedures

Although the primary lesion may be extensive or may even show distant metastases, it is always worthwhile to attempt a palliative resection, thereby improving the patient's general condition and sense of well being. This is explained by the fact that infection is reduced and anemia is corrected. In the event that it is impossible to remove the primary growth, some short-circuiting procedure, such as an esophagogastrostomy or an esophagojejunostomy may make the patient's remaining days more comfortable.

Benign Tumors

Lipomas and myomas are the most common of benign tumors which have been found in the esophagus. Owing to the constant effect of swallowing, such tumors have a tendency to become tremendously elongated and pedunculated. They have been known to be coughed up and to appear in the patient's mouth. If this occurs it may be possible to remove the lesion by means of a snare passed orally. If the tumor, however, is lower down, esophagotomy with removal of the tumor and primary closure of the esophagus is the treatment of choice.

Perforations of the Esophagus

Both spontaneous and traumatic rupture of the esophagus have been described in the literature. A spontaneous rupture is an interesting lesion and is not so extremely rare as it was formerly thought to be. If one has the condition in mind it should be possible to diagnose it preoperatively. It is found most frequently in male patients who are victims of alcoholism, and is associated with a preliminary severe bout of vomiting; this is followed by agonizing pain, which may be either thoracic or abdominal. The patient usually goes into rapid and profound shock. This condition cannot be confused with a ruptured peptic (gastric or duodenal) ulcer, since with the ruptured ulcer the pain appears first and is followed by vomiting, whereas with the spontaneous rupture vomiting comes first and is followed by pain.

The ruptured esophagus is associated frequently with surgical emphysema, which can be felt in the neck. Whenever such emphysema is noted one should think immediately of spontaneous esophageal perforation and verify this with a flat roentgen film, which will reveal a spontaneous hydropneumothorax. Aspiration of the chest has been done in some cases, resulting in the discovery of food particles or vomitus. Formerly the mortality was

thought to be 100 per cent; however, with early diagnosis and modern surgical therapy the outlook is much better today, and some patients are definitely being saved.

Mediastinitis

Perforation of the esophagus may result in a serious condition, mediastinitis. Malignant tumors or foreign bodies are the two main causes of perforation. Since carcinoma develops gradually, it does not give rise to as extensive an infection of the mediastinum as do other lesions.

Perforations caused by foreign bodies are of more practical importance. At present it is felt that mediastinal infections which extend deeper than the fourth or the fifth thoracic vertebra cannot be adequately drained by means of cervical mediastinotomy and must be treated by posterior mediastinotomy. Chemotherapy is an all-important adjunct for this type of infection.

In a presentation of this type it is impossible to mention every aspect of esophageal pathology. Only the more common and frequent conditions have been stressed. The esophagus has been just as much of a "hermit" as the pancreas; however, recently it has been brought within reach of the well equipped and well trained surgical team. That this organ is accessible to modern diagnostic and therapeutic procedures is due to the efforts of such men as Franz Torek, Sauerbruch, Garlock, Sweet, Nissen and others too numerous to mention. Their contributions have enabled the modern surgeon to save many lives that were doomed a decade ago. Today the future for esophageal surgery is most promising.

SUMMARY

The more common pathologic conditions which affect the esophagus have been discussed, and their treatments have been evaluated. The importance of physiopathology as an aid to early diagnosis has been stressed.

Since the advent of improved diagnostic methods, modern anesthesiology, chemotherapy, skillful surgical technic, and proper preoperative and postoperative care the esophagus has been brought into the realm of surgical safety.

RESUMEN

Se discuten las condiciones patológicas más comunes del esofago y se evalúa su tratamiento. La importancia de la fisiopathología como auxiliar para el diagnóstico temprano se hace notar.

Desde el advenimiento de métodos mejorados de diagnóstico, la anestesiología moderna, la quimioterapia, el desarrollo de mejor

técnica quirúrgica y los cuidados pre y postoperatorios, el tratamiento de las afecciones del esófago ha entrado en una fase de seguridad quirúrgica.

RESUME

L'auteur discute les aspects anatomiques les plus communs des affections oesophagiennes et leur traitement. Il insiste sur l'importance de la physio-pathologie, pour permettre un diagnostic précoce.

Depuis que les méthodes de diagnostic ont fait de grands progrès, que l'anesthésie s'est modernisée, que la chimiothérapie est apparue, et que les techniques chirurgicales sont plus sûres, ainsi que les soins pré et post-opératoires, on peut entreprendre avec sécurité des opérations sur l'oesophage.

Bronchiectasis in Childhood and Adult Pulmonary Tuberculosis*

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ANTONIO NAVARRETE, M.D., M.Sc., F.C.C.P.††
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The present work is based on a 15 year study of 250 cases of primary tuberculosis of childhood at the Pediatric Clinics of the Mercedes Hospital and Aballi Sanatorium for tuberculous children, at Havana, Cuba. Fifty per cent of the children included in this series were under four years of age.[1-4]

The study of these cases comprised serial bronchoscopy and bronchography with a technique appropriate to the age of the patients.[5] Bronchiectasis of varying degree was found in about 70 per cent of the cases. Reference will be made in this presentation to some demonstrative cases of bronchiectasis in chronic pulmonary tuberculosis of the adult.

It is a well established fact that bronchial obstruction, of whatever aetiology, gives rise to lobar or segmentary atelectasis and infiltration and to insufficient drainage of the bronchi, inducing alteration of their wall and retractile fibrosis. These, in turn, lead to bronchial dilatation.

Primary tuberculosis is the only common disease of childhood inducing extensive involvement of lymph nodes, of a degree sufficient to cause protracted bronchial compression and obstruction. Consequently most, if not all, bronchiectasis associated with tuberculosis in the child are caused by bronchial obstruction due to compression by enlarged lymph nodes of the primary complex, or by infiltration of the wall of the bronchi by caseous lymph nodes.

The intimate anatomical relations of the tracheo-bronquial nodes, particularly the hilar and interlobar nodes, with the bronchial branches and the small caliber of the bronchi in children, sufficiently explain these facts.

In the present series of cases bronchiectasis has been found a frequent sequel of primary tuberculosis of childhood. We are inclined to believe that systematic investigation would result in its detection in most cases of this disease, of sufficient duration.

Bronchiectasis secondary to primary tuberculosis of childhood

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has been found most ocmmonly in the right middle lobe. This fact, pointed out by Valledor several years ago, is significant in view of the condition observed occasionally in the adult and recently described under the titles of "middle lobe syndrome" or "shrunken middle lobe."[6,7]

Next in order of frequency, as the site of this type of bronchial dilatation, followed the right upper lobe, the left upper lobe, including the lingula and the lower lobes.

Bronchial obstruction by enlarged lymph nodes may be partial or complete. It is usually complete in the initial stages, when allergic aedema of the mucosa, spasm and retention of secretions act as contributory factors. It usually becomes partial after some time, but may become permanent when infiltration invades the bronchial wall followed by cicatricial stenosis.

From a diagnostic standpoint, bronchography and tomography have been found the most valuable procedures. The usefulness of bronchoscopy is limited by the fact that most lesions of the type being described affect the segmentary bronchi and thus are beyond the field of bronchoscopic visibility. Serial bronchograms are essentials in evaluating bronchographic results. During the initial stages bronchography shows a typical bronchial "stoppage" due to complete obstruction. After some days or weeks, patency is usually more or less re-established and, with penetration of the opaque medium, dilatation of the distal branches becomes evident.

The degree of dilatation gives rise to graded radiological aspects, designated the "bamboo cane," "pea pod" and "cactus" aspects, and finally to typical cylindrical, sacciform or ampular dilatations.

Bronchi in the affected zones tend to lose their normal divergence and become agglutinated, adopting the so-called "drawn fan" arrangement. In neighboring zones on the contrary, due to compensatory emphysema, the bronchial branches tend to diverge in excess and become displaced, adopting the "open fan" shape.

Clinically a large proportion of bronchiectasis resulting from primary tuberculosis of the child, particularly those of the upper lobes, are dry and do not give rise to symptoms.

Medial, lingular and lower lobe bronchiectasis are more prone to become segregating and are frequently complicated by chronic atelectasis. If extensive enough, it gives rise to displacement of neighboring structures and even to external thoracic asimmety due to retraction.

In severe cases general symptoms resulting from cardio-respiratory insufficiency supervene, with morning expectoration in increasing amounts, anoxemia and cyanosis. Haemoptisis of variable intensity, at times profuse and recurrent, is a rather frequent accident. This condition leads progressively to invalidism.

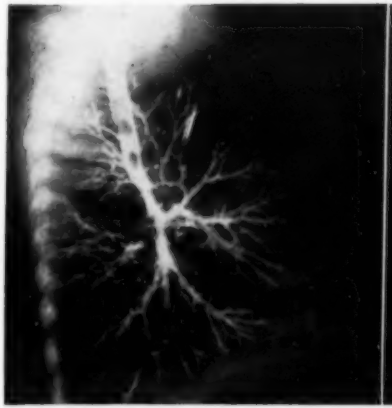

FIGURE 1

FIGURE 2

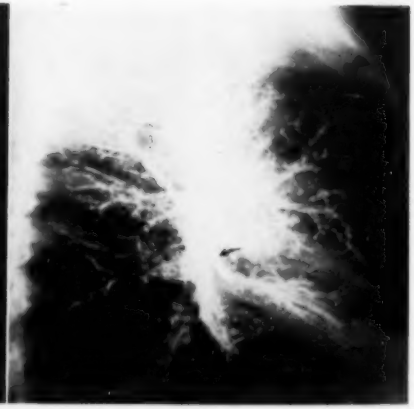

FIGURE 3

Figure 1: Normal bronchogram in a six month old child. — *Figure 2:* Z.M.C. Two and a half years old. Atelectasis of R.M.L. Stop at level of main lobar bronchus due to total obstruction. — *Figure 3*: M.B. Five years old. Atelectasis of R.M.L. Bamboo cane aspect of bronchial branches with "drawn fan" arrangement.

FIGURE 4 FIGURE 5 FIGURE 6

Figure 4: D.C. Eight years old. Chr. atelectasis of R.M.L. Partial stenosis at root of lobar bronchus. Cylindrical bronchiectasis of distal branches.—*Figure 5:* H.G. Three years old. Partial stenosis at level of root of R.M.L. bronchus. Bronchiectasis of lower branches. *Figure 6:* H.C. Two years old. Chr. atelectasis of R.M.L. Partial stenosis and dilatation of bronchi showing "pea pod" aspect. Downward displacement of branches of antero-lateral segment of R.U.L.

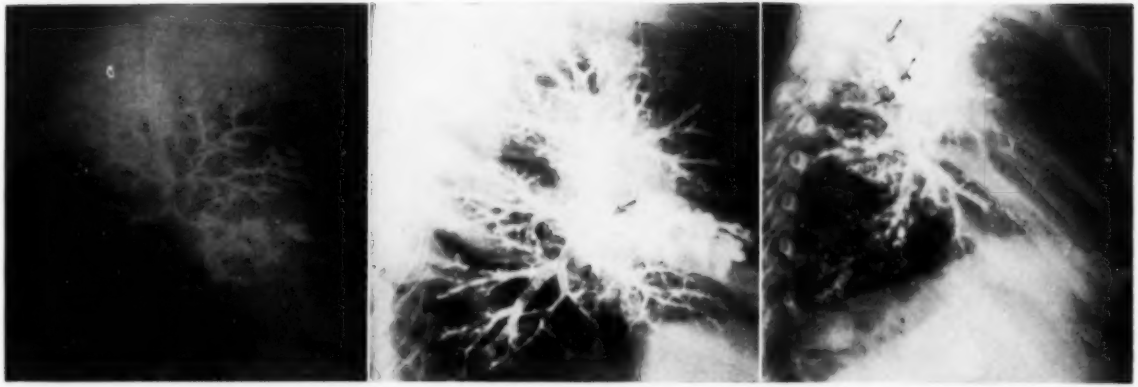

FIGURE 7 FIGURE 8 FIGURE 9

Figure 7: C.F. Four years old. Chr. atelectasis of R.M.L. Partial stenosis at root of lobar bronchus Gross distal dilatation.—*Figure 8:* J.V. Nine years old. Chr. atelectasis of R.M.L. Partial stenosis at root of lobar bronchus. Large distal dilatations. — *Figure 9:* P.M. Twelve years old. Lateral view. Partial stenosis of lobar bronchus of R.U.L. Distal bronchiectasis showing "cactus" aspect.

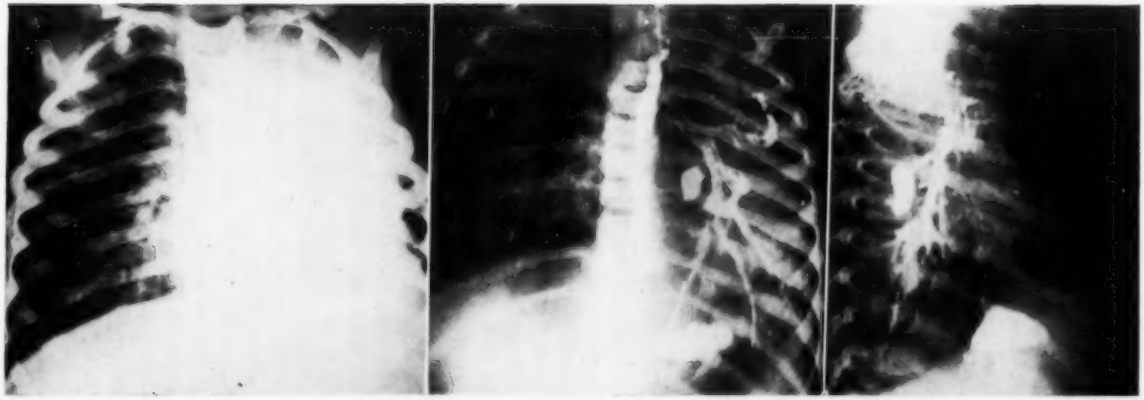

FIGURE 10 FIGURE 11 FIGURE 12

Figure 10: M.G. Three years old. Atelectasis of left lung of two years standing. Free pleural space. Increased negativity in pleura (—10 —22).—*Figure 11:* Same case as No. 9. Massive atelectasis of left lung. Dilatation and deformity of bronchi of upper lobe. Sacciform dilatation of apical or dorsal segment of lower lobe.—*Figure 12:* Same case as Nos. 9 and 10. Lateral aspect.

There is, in our minds, a strong probability that dry residual dilatations dating from the early years of life may become complicated by recurring secondary infection and lead to suppurative bronchiectasis in later life.

In the adult, bronchiectasis is a common anatomical finding in chronic pulmonary tuberculosis. The absence of large lymph node involvement and the caliber and relative rigidity of the bronchial walls, preclude the pathogenic mechanism just described in the child. Intrinsic bronchial lesions of the obstructive and stenotic type, chronic atelectasis and retraction by fibrosis are rather the pathogenic factors leading, by a well known sequence, to bronchial dilatation. These dilatations may be found in any pulmonary territory but the upper lobes are most commonly affected.

Symptoms are usually discreet and submerged clinically under those of the tuberculous process. If tuberculosis is quiescent, they may simulate activity and become a cause for concern.

Investigation of bronchial dilatation is of practical import whenever surgical treatment is contemplated or its results evaluated.[8]

SUMMARY

Bronchiectasis is a common sequel of chronic pulmonary tuberculosis, generally affecting the upper lobes.

In primary tuberculosis of infancy and childhood, bronchiectasis is the result of extrinsic compression and, at times, infiltration of the bronchi by enlarged lymph nodes. This compression leads to obstruction and atelectasis, inducing dilatation.

Segmentary branches and the main middle lobe bronchus are the ones most commonly affected by lymph node encroachment.

In most of these cases bronchiectasis is of dry uncomplicated type, but occasionally large secreting dilatation results, leading to invalidism. Recurring haemoptisis is a rather frequent symptom in such cases.

In the adult, bronchiectasis in tuberculosis results from intrinsic obstructive and stenotic lesions of the bronchi, leading to chronic atelectasis and retractile fibrosis.

There is, in all probability, a relation between residual bronchiectasis, of the type we have described in primary tuberculosis of the child and suppurative bronchiectasis in later life. This relation should be investigated by repeated examinations of patients over a long period of time.

RESUMEN

La bronquiectasia es una secuela común de la tuberculosis pulmonar crónica que afecta generalmente el lóbulo superior.

En la tuberculosis primaria de la infancia la bronquiectasia es

el resultado de la compresión extrínseca y a veces de la infiltración de los bronquios por los grandes ganglios linfáticos. Esta compresión conduce a la obstrucción y atelectasia que lleva a la dilatación.

Las ramas segmentarias y el bronquio principal del lóbulo medio son las más comunmente afectadas por la compresión ganglionar.

En la mayoría de los casos la bronquiectasia es de forma seca, no complicada pero ocasionalmente resultan grandes dilataciones secretantes que acarrean invalidez. Las hemoptisis recurrentes constituyen un síntoma frecuente en tales casos.

En el adulto la bronquiectasia en tuberculosis resulta de la obstrucción intrínseca por las lesiones obstructivas o estenosantes del bronquio que causan atelectasia crónica y fibrosis retráctil.

Muy probablemente hay relación entre la bronquiectasis residual del tipo descrito en tuberculosis primaria de la infancia y la bronquiectasis supurante de la vida adulta. Esta relación debe investigarse por repetidos exámenes del enfermo por largo tiempo.

RESUME

La dilatation des bronches est une séquelle banale de la tuberculose pulmonaire chronique. Elle atteint généralement le lobe supérieur.

Dans la primo-infection de l'enfant, la dilatation est due aux tuméfactions ganglionnaires qui agissent par compression extrinsèque et dans certains cas, par infiltration bronchique. Cette compression entraine l'obstruction et l'atélectasie qui sont elles-mêmes à l'origine de la dilatation bronchique.

Les bronches segmentaires et la bronche principale du lobe moyen sont les plus souvent affectées par l'infiltration ganglionnaire.

Dans la plupart de ces cas, les dilatations des bronches appartiennent à un type sec non compliqué, mais parfois il se constitue des dilatations largement sécrétantes, réalisant une véritable infirmité. Dans de tels cas, les hémoptysies à répétition sont un symptôme relativement fréquent.

Chez l'adulte, la dilatation des bronches dans la tuberculose est le résultat de lésions d'obstruction et de sténoses bronchiques, entrainant une atélectasie chronique et une sclérose rétractile.

Il y a toute probabilité pour que les dilatations des bronches avec suppuration qui peuvent survenir plus tard soient en relation avec la forme que nous avons écrite dans la tuberculose primaire de l'enfant. Cette relation devrait être recherchée par des examens répétés pendant une très longue période de temps.

REFERENCES

1 Valledor T.: "Localizaciones Lobares y Segmentarias de la Tuberculosis de Primo-infecci n en el Niño. Pneumonitis Obstructiva y sus

Secuelas," *Rev. Cubana de Pediatría*, 19:189, 1947.
2 Valledor T.: "Las Bronquiectasias Secundarias a la Tuberculosis Primaria de la Infancia," *Rev. Española de Pediatría*, III:1:39, 1947.
3 Valledor T. y Cantlon B.: "Factor Bronquial y Tuberculosis de Primoinfecci n en el Niño," *Rev. Cubana de Pediatría*, 20:3, 1948.
4 Valledor T. y colaboradores: "El Factor Bronquial en la Patología Respiratoria del Niño. Las Bronquiectasias y sus Secuelas," *Rev. Chilena de Pediatría*, 18:955, 1947.
5 Rouco J. M.: "Broncografía en los Niños Pequeños," *Rev. Cubana de Pediatría*, 19:666, 1947.
6 Rubin, Eli H. and Rubin, Morris: "The Shrunken Right Middle Lobe," *Dis. of Chest*, 18:127, 1950.
7 Graham, E. A., Budfor, T. H. and Mayer, J. H.: "Middle Lobe Syndrome," *Postgraduate Med.*, 4:29, 1948 (cited by 6).
8 Navarrete A. y Abreu J. P.: Comunicaci n a la Sociedad Cubana de Tisiología, Habana, December 1943.
9 Navarrete A.: "Contribuci n a la Sistematizaci n del Tratamiento de las Cavidades Residuales. De la Ponencia Sistemática del Tratamiento de las Cavernas del VI Congreso Panamericano de la Tuberculosis," *Editorial Neptuno*, Habana, January 1945.

Pulmonary Coccidioidomycosis

Clinical and Roentgenological Observations in Residual Lesions*

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Human infection with coccidioides immitis was first described by Posadas in Argentine in 1892.[1] Rixford and Gilchrist reported in 1896 the first two cases on the North American continent, and named the organism coccidioides immitis.[2] In 1937, Dickson and Gifford described the benign, primary type of infection.[3] The intricate blood serum studies with reference to complement fixation and precipitins have been developed by Smith.[4]

Persons showing residuals of the primary infection are now observed in every part of the United States. This is due to the World War II period during which time hundreds of thousands of troops were stationed for training purposes in the endemic regions, and made possible exposure to and infection of these individuals by coccidioides immitis Of especial interest to us is the experience that latent, apparently innocuous, residual lesions of the primary infection may reactivate, producing focal or disseminated pathological activity.

Coccidioides immitis also causes disease in cattle, sheep, and dogs.[5] Emmons (1943)[6] found rodents infected as well, and suggests that the disease may be primarily one of rodents, and that as the result of contamination of the soil by their excreta, the disease is propogated to man. The disease is dust borne, and the infection rate is highest in the dry summer and autumn seasons. Coccidioides immitis has been cultured from the soil. Smith et al. have conducted experiments on infection control among air corps personnel stationed in the arid regions of the Southwest. Paving roads and runways, spraying oil on athletic fields and planting lawns, reduced considerably the incidence of infection.

Comparable to the epidemiologic experience on the part of the

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native population in an endemic area,[7] our troops experienced little or no clinical illness or physical disability following a variable period of exposure to the fungus. The service records indicate that for the most part, cases showing coccidioidal residual since leaving the training area, did not seek medical attention during the period of exposure. According to Smith 25 per cent of the military personnel became infected. A good number, however, became ill and were hospitalized. Fortunately, the total number that succumbed to the infection was small. It occurred when the disease assumed a disseminated character.

Pathogenesis: Regarding the development of primary coccidioidomycosis, Smith has postulated the following: "In the asymptomatic form of infection without roentgenologic findings, the lesion can be assumed to be too small to be detected by x-ray. This is the type of infection which shows a positive skin test, and is the one which affects the majority of the residents in an endemic area. In the more severe form, characterized by pneumonitis, the symptomatology reaches a peak at about ten to fourteen days following infection, accompanied by sensitization to the fungus as evidenced by a reaction to coccidioidin. A positive skin test appears about 10 to 45 days after the infection is acquired. Shortly after the infection is established, precipitins and fixation of complement may be detected. In the usual course of events, the coccidioidal infection is walled off, presumably as in tuberculosis. The primary focus undergoes fibrosis and frequently calcifies. As in tuberculosis, the person retains his allergy" Regarding the duration of this allergy, Smith has been able to trace people who left an endemic area as long as 30 years before and are still able to react vigorously to coccidioidin.

Clinical, Pathological, and Roentgenological Considerations in Primary Coccidioidomycosis. Primary coccidioidal infection is classified by Smith as follows: 1) The primary infection, and 2) The disseminated form, commonly designated as coccidioidal granuloma. The primary infection group is further classified into the following: (a) The asymptomatic form, (b) the symptomatic form, and (c) the cavitory form.

The asymptomatic form of the infection was outlined in the paragraph on pathogenesis. The symptomatic form of the disease, designated as the influenzal or pneumonic type, is characterized by acute illness, accompanied by malaise, fever, cough, chills, dyspnea, chest pain, increased sedimentation rate, and elevated eosinophil count. This type of infection was responsible for the admission of our troops to the hospital. The cavitary form of coccidioidal infection results from a necrotizing process occurring within the area of pneumonitis. Spontaneous closure of this cavity

is a common phenomenon. In some instances, however, it fails to close, and is seen to persist for many years despite recovery from acute illness and subsequent well-being. The potential hazzard inherent in this cavity has already been stated.

The serious form of the disease is the coccidioidal granuloma. Smith estimates its incidence to be once in 1000 to 5000 persons infected. It shows a predilection for dark-skinned persons, such as the Negro, Filipine, and the Mexican.

The most common roentgenological findings during the acute phases of the infection are the following: 1) pneumonic densities; 2) hilar and/or mediastinal lymphadenopathy; 3) pleural effusion, and 4) cavity formation. Carter[8] states that during the acute phase of the lung invasion the following differential diagnosis should be made: Lymphomatous diseases, sarcoidosis, and carcinoma, as well as blastomycosis, actinomycosis, and moniliasis, ect. Tuberculosis is not stressed in differential diagnosis. We feel that it should be added for completeness.

Residual Lesions: The residual lesions of the infection with coccidioides immitis are observed most commonly in the medical clinics of the Veterans Administration. The present communication concerns a group of ex-service men. The observation period extends from February 1946, to February 1950. This study concerned an analysis of their physical status, sensitivity to coccidioidin, the character of the roentgenological residuals, and the character of the reactivated lesion. Mycologic study to isolate the fungus coccidioides immitis was not performed routinely, but was reserved for those individual cases that were suspected of having active pathology, or were going through a reactivation of the latent focus.

The most common residual is the nodular density, consisting of a single, small, fibrous infiltration, complemented by a positive skin test to coccidioidin. Our observations indicate that these nodules may be multiple. Moreover, instead of the well-circumscribed nodular density, there may be present single or multiple large masses, with little or no visible fibrous reaction around them. We consider these as representing the granuloma of latent coccidioidomycosis, similar to a tuberculoma. Each is equally capable of spontaneous dissemination. This phenomen was observed in one of our cases.

A number of observers have stressed the presence of calcium within or adjacent to these nodules. Sweigert et al.[9] state that they have observed rather frequently a central calcific stippling within the nodule. A similar observation is made by Berke, Schomer, and Bass,[10] who in a recent report on coccidioidomycosis residuals describe a case with cavity and neighboring small calcific deposits. In our series of cases no calcification was observed

within the nodular residuals, nor have we noted calcium deposition in the tissues adjacent to these or cavitary lesions.

An organized effusion is another type of residual which is the end-result of an effusion that accompanies the pneumonic form of a primary infection. This occurred in some of the cases reported by Bass et al.,[11] and Taylor and Briney.[12] Our case of coccidioidal granuloma showed a concomitant organized effusion for several years.

Other, less common, residuals are: Miliary lung infiltration, chronic pneumonitis, and mediastinal and hilar adenopathy. In our series we encountered one case of chronic pneumonitis that accompanied a residual cavity.

The coccidioidal cavity residual is of arresting interest. Considerable controversy exists regarding its development, its mutability, and mechanics responsible for closure. The anatomical characteristic is the thinness of its wall. Some observers have stated that it resembles a tuberculous cavity We wish to point out that it resembles much more closely a thin-wall cyst.

Sometimes, residual coccidioidal cavitation is multiple. One of our cases presented multiple cavitation on the right and a single cavity on the left. In discussing the problem of therapy of cavitary lesions, Krapin et al.[13] report such a lesion of an upper lobe treated by lobectomy. Following surgery, three cavities made their appearance in the lower lobe. Their opinion is that latent foci of disease in the lower lobe became reactivated following mobilization and hyper-expansion of the lower lobe, resulting in development of cavity.

Regarding the relative frequency of nodular residual as compared with the cavitary variety, Berke et al. find that the former is observed more frequently than the latter. Our findings confirm the observations of these writers.

Regarding tissue sensitivity, all of our cases, except one, showed the conventional 2 plus reaction to coccidioidin. Tissue sensitivity can be of a marked degree. This was observed in one of our cases. The introduction of 0.1 cc. of the 1:100 dilution antigen induced anaphylactic shock necessitating repeated doses of adrenalin to bring about recovery. The case presenting this phenomenon of sensitivity showed roentgenologically a benign nodular coccidioidal residual.

Analysis of Material: In our clinic 23 cases of coccidioides immitis infection residuals were observed over a four year period. All of the cases were in good health, except when a reactivation had taken place, and a considerable period of exhaustion followed pathological activity. With the exception of one case already cited, our cases showed 2 plus reaction to coccidioidin. Roentgenolog-

ically, 12 cases showed a nodular type of residual; seven a cavitary residual; and four no visual residual One showed multiple nodules. One showed multiple granuloma with very little fibrotic encopsulation, and concomitant organized pleural exudate. One of our cases had multiple cavities, which were bilateral. One showed chronic pneumonitis as a concomitant of a cavitary residual. We looked for residuals such as miliary densities, hilar adenopathy, and calcific stippling within the nodular densities as well as within the parenchyma adjacent to the cavity. We observed only one of these entities, namely, hilum adenopathy (Case 2).

Mycologic study revealed the following: One apparently latent, inactive, cavitary case showed coccidioides immitis spherules on culture. The second case presented the spherules during the reactivation phase and several months later. A third case with cavitary disease accompanied by profuse hemorrhage, showed the spherules of coccidioides immitis within giant cells in sections of the excised lobe. A fourth case, exhibiting bilateral cavitation, and severe hemoptysis, had positive sputum cultures before and after surgery. A fifth case with dissemination of the disease from the lungs to the lymph nodes of the neck, showed coccidioides immitis spherules in the purulent material obtained from the draining sinuses, and also in the material of the excised nodes.

Reactivation of Latent or Residual Coccidioidomycosis Lesions:

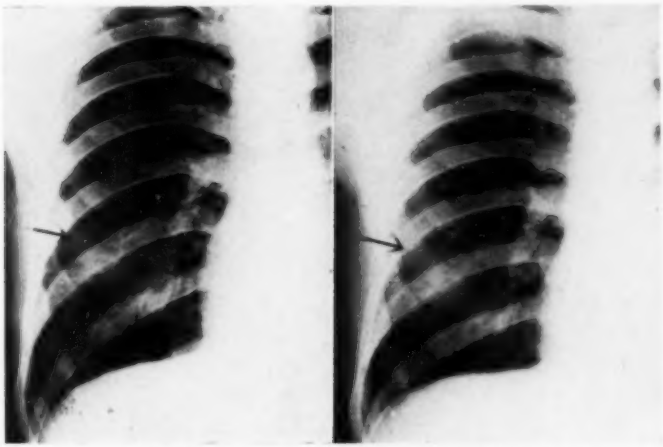

FIGURE 1 FIGURE 2

Figure 1, Case 1: December, 1948. Fibrous infiltration surrounding a thin-wall cavity, 2 x 4 cm. (arrow), observed six weeks following focal reactivation of cavitary residual. — *Figure 2, Case 1:* October, 1949. Marked improvement. Cavity now measures 1.5 x 1.5 cm.

The majority of reported cases deal with pathological activation of cavitary lesions. Hemorrhage and/or pneumonitis are the most common phenomena. However, in cases in which dissemination is suspected, pathologic phenomena will be looked for in other organs, involving the lymphatic, osseous, skin, and nervous systems. In a report from a Veterans Center,[11] one out of four cavitary residual cases had a lymph-hematogenous extension involving the lip and nose in the form of cutaneous lesions.

In our series of cases studied there occurred reactivation in four cases. Of these, three had developed pathological activity in the presence of cavitary residual. In the fourth case, reactivation occurred in the presence of massive, nodular, residual. The first three cases had focal reactivation, and each was characterized by hemoptysis. In the fourth, the reactivation was accompanied by dissemination of the residual, and by a process of lymph-hematogenous extension involving the cervical chain of lymph nodes. It is of especial interest that the latter case was considered serologically within the dissemination range of the infection for which he was hospitalized in New Mexico one year and five months previously.

Case 1. (Latent cavitary residual accompanied by focal reactivation characterized by hemoptysis and pneumonitis). Military History: In-

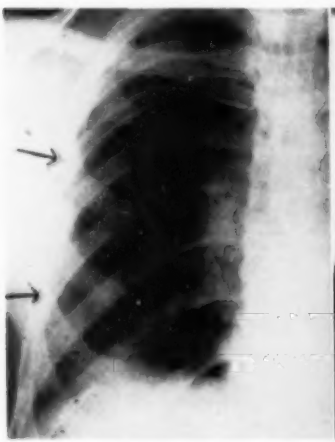

FIGURE 3

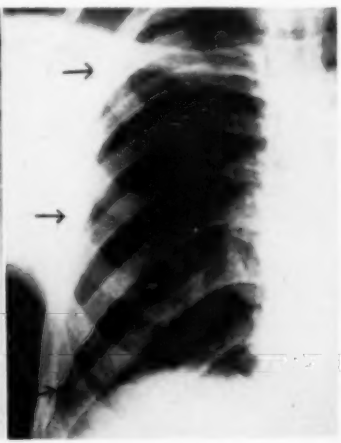

FIGURE 4

Figure 3, Case 2: March, 1948. A number of coccidioidal infection residuals are present: Hilum adenopathy, multiple granuloma (arrows), thickened pleura, and infiltration above diaphragm. Dissemination took place.—*Figure 4, Case 2:* January, 1950. Granulomatous masses nearly gone; three thin-walled cavities visualized as new lesions at costophrenic zone; pleural reaction markedly diminished; hilum adenopathy shows marked recession.

ducted in Boston, October 1942. After three months of local basic training transferred to Arizona in January 1943. After three months was transferred to San Joaquin Valley in California where he remained until he was discharged from the service in February 1946. Entire tour of duty was uneventful except for a continuous cough which began in Arizona and persisted till he had a reactivation in April 1948, two years and two months after discharge. He was well and working till he became acutely ill the last week of April 1948, when he developed increasing cough, sputum became profuse, and it was accompanied by frequent hemoptysis, fever, and chest pain. He entered the emergency ward of the Boston City Hospital where he did not react to tuberculin (dilution 1:10,000) but reacted to a dilution of 1:100. Coccidioidin antigen showed a 2 plus reaction. Blood serum studies performed by Dr. Smith of Stanford University revealed a negative test for precipitins, while the fixation of complement was an indication of an active coccidioides immitis infection. Spherules of coccidioides immitis were found in sputum by direct and culture methods. X-ray films revealed evidence of a cavitary lesion with a zone of pneumonitis around it. Following discharge from the hospital he reported to the Regional Office. A survey revealed the following: Induction x-ray films of chest were negative. Separation x-ray films showed evidence of a cavitary lesion surrounded by a zone of pneumonitis; this was comparable to the pathology that was found after hospital discharge. Obviously this case illustrates a silent coccidioidal residual, resulting from an infection acquired while in the endemic area, and manifested by cough which began soon after exposure. Finally pathological reactivation occurred characterized by hemoptysis and fever. Follow-up observation shows continuous regression of the pneumonitis and shrinkage of the cavity.

FIGURE 5

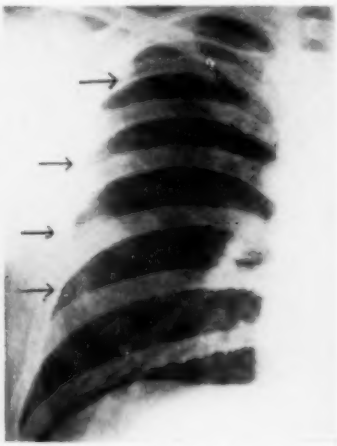

FIGURE 6

Figure 5, Case 3: January, 1947. Single, well circumscribed, nodule, 2 cm. diameter; infection occurred in 1942.—*Figure 6, Case 4:* October, 1947. Coccidioidal residual consisting of four nodules (arrows). No change during a four year observation period.

Case 2. (Case showing dissemination). This Negro, age 25, was inducted in Boston, April 1945. He arrived in Texas in May 1945 and remained there till September 1945. He was well until September 25, 1945, when he became acutely ill. At the station hospital he showed, in succession, generalized itchy eruption, severe right sided chest pain and fever of 100 to 102 degrees F. X-ray film showed evidence of pneumonitis with effusion at right base. W.B.C. 20,000; eosinophils 70 per cent; R.B.C. normal. By November, W.B.C. fell to 12,400, and eosinophils dropped to 1 per cent. Sedimentation rate between November 10th, and December 31st ranged between 65 mm. and 92 mm. By January 2, 1946, the sedimentation rate dropped to 18 mm./hour, W.B.C. was 13,100, and eosinophils 2 per cent. At this time, findings for heterofil, tuloremia, tuberculosis, and bacteriemia were negative, but the skin test with coccidioidin was positive. No clinical benefit was obtained from sulfa and penicillin. By January 25, 1946, he was in a general hospital for further study when the W.B.C. was 11,700, eosinophils 3 per cent and sedimentation rate rose to 56 mm./hour. Three gastric washings during February and March, were negative for coccidioides immitis, but a gastric smear in May showed a few spherules of coccidioides immitis. A gastric in June showed rocket-shaped mycelial organisms resembling coccidioides immitis. By July 1946, x-ray films showed a nodular infiltration in the right subapical region, and pneumonitis and fluid still in the base. Fixation of complement and titer of precipitins were positive to 4 plus in all significant dilutions. Dr. Smith's conclusions were: "This Negro has really a severe coccidioidal infection. Titer is in the range of dissemnation. Advise he continue bed rest." In two subsequent analyses, a complement fixation titer rise (dissemination still present) in one, and a definite

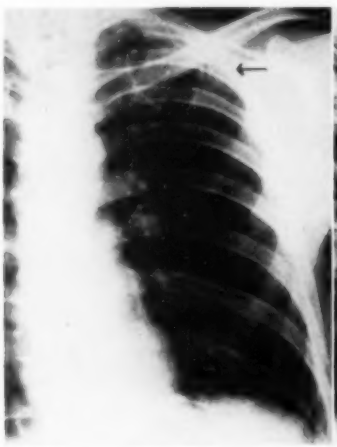

FIGURE 7

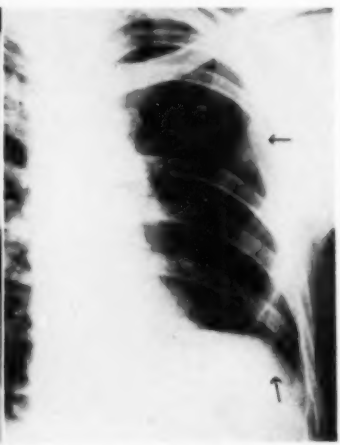

FIGURE 8

Figure 7, Case 5: January, 1949. Thin-walled apical cavity, 5 x 6 cm. Note absence of reaction around cavity. — *Figure 8, Case 5:* One month later: Lobectomy performed (upper arrow) and diaphragm elevated as result of phrenic crush (lower arrow).

fall in complement fixation titer in the other (indicating real improvement) were noted. He was discharged from service on August 13, 1946, and was apparently well until admission to hte West Roxbury Veterans Hospital. The history was as follows: During the previous year he noted swelling and redness in the neck under the chin. Incision and drainage was done, and the sinus closed after one month. Three months before admission, swelling recurred, and it was again incised, and a sinus was established. The hospital examination confirmed the previous findings, and at this time a large mass was also found on the right side of the neck. The W.B.C. was 10,200 and differential was normal. An excised lymph node showed coccidioides immitis on culture. A complete excision of the neck masses was done. X-ray films revealed a zone of density at the right base. He made an uneventful recovery. By January 1950, the pulmonary lesion showed remarkable mutation: There are now present three cavitary lesion shadows, and the granulomatous masses seen previously are not visualized. His general condition is excellent (October 1950).

Case 3. (Single nodular residual). This man had three months training period in the Southwest in 1942. No symptoms appeared until January 1944 when he was stationed in India and had a four day period of "flu". In June 1945, he had a week's illness consisting of malaise, fever, and cough (India). He was separated from service in September 1945, when a nodular density was noted. He has since been in good health and x-ray films show no change (1950). The coccidioidin test is positive.

Case 4. (Multiple nodular residual). This man had four months desert training in 1945. At the end of that time he was hospitalized because of cough, fever, and malaise of three weeks duration. The coccidioidin test was positive. He made an excellent recovery with no reactivations to date (1950).

Case 5. (Hemorrhagic reactivation of cavitary lesion). This man had extensive duty in desert areas of Texas and California. In April 1945, a discharge x-ray film showed evidence of a large cavity in left upper lobe. No symptoms while in Southwest. On separation from service coccidioides immitis spherules were found in sputum culture. Studies were negative for tuberculosis. He was first seen at the West Roxbury Veterans Administration Hospital in June 1946 when he had no symptoms, and x-ray shadows were unchanged. In August 1946, there was questionable progression, but he was asymptomatic. Bed rest at home was advised. By January 1947, cough had increased, and he had blood streaked sputum at intervals. In December 1947, because of intermittent hemoptyses, he was admitted for surgical observation. A phrenic crush was done, and he soon went home. During the next year he was well, but had repeated hemoptyses. In January 1949, lobectomy was performed. The phrenic nerve was re-crushed in February. One year later he was asymptomatic and was leading a fairly normal life. No residual or new pathology was seen.

Case 6. (Bilateral multiple cavitary residuals with severe hemorrhagic reactivation). This man was stationed in Arizona for several months in 1943. There was no history of respiratory illness untill March 1946, when he noted cough, sputum, and blood streaks. X-ray films showed evidence of thin-walled cavities. Symptoms were unchanged till May 1949,

FIGURE 9

FIGURE 10

Figure 9 ,Case 6: May, 1949. Four thin-walled cavities in the right upper lobe, the largest measures 5.5 x 6.5 cms. No pericavitory reaction. Note cavity in left mid-lung field, 1.0 cm. size.—*Figure 10, Case 6:* January, 1950. Following excision of right upper lobe and thoracoplasty. Thin-walled cavities are noted in left lung; the cavity in mid-field has increased to 1.5 cm.

when he began to have large hemoptyses. Coccidioides spherules were found in sputum on culture. On July 21, 1949, the right upper lobe and the superior segment of the right lower lobe were resected. He made a good recovery, but positive cultures persisted. Follow-up has shown enlargement of a contralateral left upper lobe cavity, and two new cavities have appeared in the left lower lobe. Partial thoracoplasty (second, third and fourth ribs) was done to supplement the lobectomy. To date (December 1950) he continues at bed rest at home.

Comment

Considered in past years a serious disease with an expected fatal evolution, coccidioidomycosis was regarded as the dread disease of the Southwest until it became known that a mild form of the infection prevailed. Knowledge concerning the disease was later implemented by mycologic studies which demonstrated the presence of coccidioides immitis "spherules," or the parasitic form of the fungus, in cultures of material from coccidioidal lesions Thus developed a fuller understanding of the epidemiology of the disease, when it became known that the saprophitic form of the organism, the clamydospore, is confined to the soil of the endemic area, whereas the sporangia, or "spherules," are the parasitic elements of the organism found in the infected human. Later observations pointed to the phenomen of antigenicity, when it became known that within days or weeks following infection, the person developed a positive skin test to the coccidioidin antigen. A corollary to the positive coccidioidin skin test is the occurrence of erythema nodosum or erythema multiforme in newly infected persons. The incidence of this manifestation is approximately 2 to 5 per cent, and is considered due to tissue sensitivity initiated by the presence of circulating coccidioidal antigen.

In the main, the disease is characterized by two principal forms: 1) The influenzal or pneumonic type, and 2) The disseminated form, or coccidioidal granuloma. The former has its pathological manifestations focalized in the thorax, whereas the latter shows invasion of the skin, the osseous and nervous systems, having metastasized by way of lymph-hematogenous channels from the lungs.

The latent, or inactive, residual foci of infection with coccidioides immitis have a variety of roentgenological findings, and are potentially capable of pathological reactivation, and/or anatomical mutation. The roentgenological elements observed by us were of the following nature: cavity with surrounding pneumonitis; cavity without pneumonitis, both single and multiple, coccidioidal nodule, single and multiple; coccidioidal mass or granuloma. Regarding the manifestations of reactivated latent lesions, two particular phenomena were noted: Hemorrhage as part of a focal reactiva-

tion, and dissemination by way of the lymphatic system with involvement of the corresponding lymph nodes. A third phenomenon observed was an anatomical mutation in the case presenting dissemination. In this case the initial granuloma masses finally changed into a cavitary form residual.

SUMMARY

1) Because of the exposure of large numbers of ex-service people to the endemic Southwest during the recent war years, coccidioidal residuals are now observed in every part of the country.

2) Though the vast majority of cases harboring coccidioidal residuals are maintaining a good health status, a certain proportion have already experienced pathological reactivation in the past, and are expected to reactivate at any time in the future.

3) Illustrative cases are presented in this paper, dealing with the problem of coccidioidal cavity necrosis and hemorrhage, as well as the operative treatment by means of lobectomy of those cases that presented intractable hemorrhage.

4) It is our opinion that in spite of the comparatively low incidence of the disseminated form of coccidioidal residual reactivation, it is an ever-present danger, and may prove fatal.

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RESUMEN

1) Debido a la exposición de gran número de personas que estuvieron en servicio militar en el Suroeste durante los años de la reciente guerra, los residuos de la coccidioidomycosis se observan en todas partes en el País.

2) Aunque la gran mayoría de los casos que tienen esos residuos se encuentran en buena salud, ciertos de ellos han experimentado reactivación patológica en el pasado y se espera que se reactiven en el futuro.

3) Se presentan casos ejemplares en este trabajo tratando el problema de la necrosis cavitaria de coccidioisis y la hemorragia así como el tratamiento operatorio por la lobectomía en los casos de hemorragia no dominable.

4) Es nuestra opinión que a pesar de la incidencia comparativamente baja de la forma diseminada de residuos de coccidioido-

micosis reactivada, es esta siempre un peligro presente que puede ser fatal.

RESUME

1) Un grand nombre de mobilisés au cours de la récente guerre furent exposés à la coccidioidomycose endémique qui règne dans le Sud-Ouest. Ceci explique que des séquelles de coccidioidomycose soient maintenant observées un peu partout dans le pays

2) Bien que l'immense majorité de ces malades atteints de séquelles de coccidioidomycose soit en bonne santé, il faut noter une certaine proportion de récidives déjà apparues, et il faut craindre que dans l'avenir les malades en puissent être l'objet.

3) Les auteurs rapportent des cas caractéristiques. Ils soulèvent à leur sujet le problème des ulcérations nécrotiques et des hémorragies dues à cette affection. Ils exposent le traitement opératoire par lobectomie dans les cas qui se sont compliqués d'hémorragies intarisables.

4) Leur avis est que, malgré le peu d'importance relative des réactivations des séquelles de coccidioidomycose, les malades sont menacés d'un danger toujours présent, et qui peut devenir fatal.

REFERENCES

1 Posadas, A.: "Un Nuevo Caso DeMicosis Fungoidea con Psorospermias," *Am. Circ. Med. Argentine*, 15:585, 1892.
2 Rixford, E. and Gilchrist, T. C.: "Two Cases of Protozoan (Coccidioidal) Infection of the Skin and Other Organs," *Johns Hopkins Hospital Reports*, 1:209, 1896.
3 Dickson, E. C. and Gifford, M. A.: "Coccidioides Infection (Coccidioidomycosis), II. The Primary Type of Infection," *Arch. Int. Med.*, 62:853, 1938.
4 Smith, C. E.: "Coccidioidomycosis," *Med. Clin. North America*, 27:790, 1943.
5 Stiles, G. W. and Davis, C. L.: "Coccidioidal Granuloma (Coccidioidomycosis): Its Incidence in Man and Animals and Its Diagnosis in Animals," *J. Am. Med. Assn.*, 119:765, 1942.
6 Emmons, C. W.: "Coccidioidomycosis," *Bull. Hyg.*, 18:475, June 1943 (Abstract).
7 Randolph, Howell and McMartin, H. L.: "Coccidioidomycosis in Phoenix, Arizona," *Dis. of Chest*, 13:471, 1947.
8 Carter, R. A.: "Roentgen Diagnosis of Fungus Infections of the Lungs with Special Reference to Coccidioidomycosis," *Radiology*, 38:649, 1942.
9 Sweigert, C. F., Turner, J. W. and Gillespie, J. B.: "Clinical and Roentgenological Aspects of Coccidioidomycosis," *Radiology*, 38:663, 1942.
10 Berke, Rudolph, Schomer, Alexander and Bass, H. E.: "Residual Cavities in Coccidioidomycosis: Follow-up Studies," *Dis. of Chest*, 17:84, 1950.
11 Kurz, Ed. R. H. and Loud, Norman W.: "Coccidioidomycosis in New England," *New Eng. J. Med.*, 237:610, 1947.

Pulmonary Cavitation with Negative Sputum

(With Reports of Eight Cases)

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On most general medical services and in the office of the internist, the appearance of pulmonary cavitation on x-ray film, particularly in the upper lobes, is usually diagnosed as tuberculosis, even in the absence of positive sputum. In the majority of cases this diagnosis proves to be correct, only because tuberculosis is more common than the other pulmonary diseases causing cavitation and not because the cavity is in any way specific for tuberculosis.

Cavitation itself is merely a general expression of lung pathology, and this paper aims to restate this fact and to review the other lung lesions which are too frequently admitted to a tuberculosis service with an erroneous diagnosis.

Consideration of the mechanism of cavity formation in the lung makes it apparent why this particular type of lesion is specific, not for any one etiological agent, but for the lung tissue in which it is found.

Carylios[1] pointed out that, once the continuity of lung parenchyma is interrupted by caseation or any other type of tissue destruction, there is an immediate tendency for cavity formation. The defect does not collapse as it would in any other organ, because the negative intrapleural pressure, together with the atmospheric pressure in the communicating bronchus serve to keep the cavity open and spherical as would fluid under pressure in another organ.

Andrus[2,3] felt that the difference in intracavitary pressure above or below that of the surrounding lung, was a result, rather than a cause, of the elastic pull of the lung and he attributed cavity formation to the elastic tension on pulmonary tissues, the continuity of which had been interrupted by any disease process. He postulated an abnormally high elastic tension in diseased tissues bordering on areas of atelectasis. The importance of the elastic tissue in the formation of spherical cavities was illustrated mechanically by Reinders[4] who demonstrated that small, irregular holes burned into an elastic membrane became perfectly round when that membrane was subjected to stretch. Moolten[5] produced cavities in isolated human and calf lungs by lacerating the bronchi

and surrounding lung tissue with bronchoscopic punch forceps and then inflating the lungs; these cavities had the gross and roentgen appearance of lesions produced by disease and proved that it is the mechanical and physiological nature of the lungs, rather than any specific bacilli, which accounts for pulmonary cavitation. Any etiological factor causing lung destruction can therefore cause cavitation.

The more common cavity-producing, non-tuberculous lesions which are admitted frequently to tuberculosis hospitals because of misleading x-ray film appearance, may be classified as follows:

I. Infectious lesions
   - A. Resolving pneumonia
   - B. Lung abscess
   - C. Fungus infection
     1. Coccidioidomycosis
     2. Histoplasmosis
     3. Aspergillosis
     4. Actinomycosis
     5. Other fungi

II. Non-infectious
   - A. Cystic disease of the lung
   - B. Neoplasm
     1. Bronchogenic carcinoma
     2. Metastatic neoplasm
   - C. Pulmonary infarction
     1. Secondary to emboli
     2. Polyarteritis
   - D. Trauma.

Resolving Pneumonia: As pointed out by Cohen,[6] pneumonia, particularly during the stage of resolution, may simulate tuberculosis in the roentgenogram. In these cases the large cavities seen on x-ray film are due, not to extensive tissue destruction, but either to zones of clearing within an area of greater density, or to ballooning of a few alveoli secondary to inflammatory obstruction of the bronchus with neighboring atelectasis. The latter process is similar in mechanism to true cavity formation.

In differentiating these cases from tuberculosis, acuteness of onset, absence of tubercle bacilli from the sputum, and particularly rapidity of response to treatment, suffice to make the diagnosis. In Friedlander's pneumonia where the appearance of cavitation on x-ray film may actually be due to multiple small lung abscesses and where there may be a slow response to streptomycin as in pulmonary tuberculosis, culture of the organism from the sputum at the initiation of treatment will be diagnostic.

Cases 1 and 2 illustrate the not uncommon error of diagnosing pneumonia as pulmonary tuberculosis on the basis of the x-ray picture alone:

Case 1: F.P.S., a 24 year old white male was admitted to the hospital on November 1, 1941 with a history of fever, anorexia, malaise and cough for five days, following an asthmatic attack. On October 27th he noted a dull aching pain in the left anterior chest, and chilly sensations. On admission, physical examination revealed a weak, toxic young man with increased conduction of breath and voice sounds in the left apical region. Scattered musical rales were noted.

X-ray and laboratory findings: X-ray film inspection on November 1st showed soft homogeneous density in the upper third, left lung. On November 9th there was some clearing with cavitation under the first rib. On November 21st resolution had continued. On November 23rd the chest appeared clear except for a few "streaks" in the left apex.

One sputum was positive for acid-fast organisms. However, this was probably a false report, as subsequently sputum was negative on 16 tests by smear, culture, and guinea pig inoculation. Three gastric washings were negative. Tuberculin test was repeatedly negative (P.P.D. both strengths).

Course: The patient was first diagnosed as having pneumonia, but when he failed to respond dramatically to chemotherapy, when one sputum was reported positive for acid-fast bacilli, and when a cavity developed in the left apex, the diagnosis was changed to tuberculosis. However, the chest cleared almost completely within two months, repeated tuberculin tests and further sputum studies were negative, and a final diagnosis of bronchopneumonia, non-specific type, probably virus, was made. The patient gained 12 pounds and made a complete recovery.

Case 2: H.W., a 22 year old white male was admitted to the hospital on May 31, 1941. About May 1st he had developed a "cold" with pain over the right breast and fever. He was admitted to a station hospital May 15th

FIGURE 1a FIGURE 1b

Figure 1a, Case 1, F.P.S.: Pneumonia left upper lobe.
Figure 1b: Same patient six weeks later, showing complete clearing.

where all symptoms cleared after a few days. Sputum was negative for tubercle bacilli, but an x-ray film taken May 15th showed an infiltration of the right upper lobe with a 1.5 cm. cavity. A diagnosis of tuberculosis was made and he was transferred with a recommendation that artificial pneumothorax be instituted. Physical examination on admission was negative.

Laboratory and x-ray findings: An x-ray film of the chest on June 2nd showed the lesion seen on May 15th to have cleared completely. Sputum was negative on five tests on smear, culture and guinea pig inoculation. Tuberculin skin test was negative (P.P.D. both strengths). He was observed for 30 days and then discharged with a diagnosis of bronchopneumonia, atypical, right upper lobe.

Lung Abscess: Despite the presence of cavitation, lung abscess is usually differentiated without difficulty from tuberculosis on the basis of the history, the more septic course, the expectoration of foul sputum, and the absence of tubercle bacilli in the presence of a large cavity. The occurrence of lung abscess secondary to fungus infections, bronchogenic carcinoma, or septic infarctions, will be discussed under those headings. Sellor and his co-workers[7] have also described a syndrome of spreading suppurative pneumonitis, a non-specific progressive consolidation of the lung with extensive abscess formation which may closely mimic tuberculosis.

Fungus Infections: Hobby[8] in summarizing the many fungi that can produce pulmonary disease lists coccidioidomycosis, aspergillosis, histoplasmosis, geotrichum, and torulosis as the cavity-producing fungi. Until recently it was believed that the pulmonary lesion of histoplasmosis was a miliary infiltration with ultimate diffuse calcification, but a recent study by Furculow[9] showed that of 16 cases, four showed cavitation with x-ray pictures that, in the absence of laboratory work, would appear identical to tuberculosis. Chortis[10] emphasizes the predilection of aspergillosis for

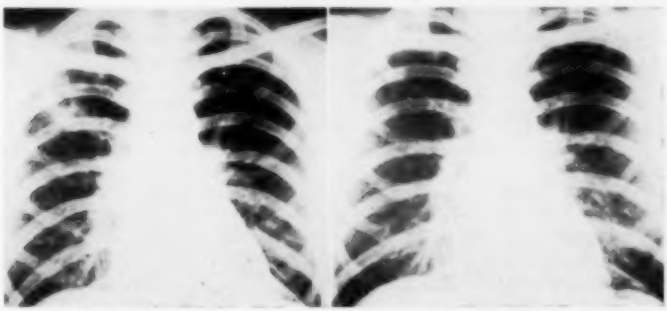

FIGURE 2a FIGURE 2b

Figure 2a, Case 2, H.W.: Pneumonia right upper lobe with cavity.
Figure 2b: Same patient, complete clearing in two weeks.

the upper lobes and apices of the lungs, with cavitation. Cavitation is also a commonplace in coccidioidomycosis. However, Smith and his group[11] and Berke and co-workers[12] have found that since cavitation in coccidioidomycosis may occur as a late sequel to a previous silent infection, appearing long after the patient has left the endemic area, the original etiologic agent may easily be missed. Krapin and Lovelock[13] have also reported a case in which, following right upper and middle lobectomy for a bleeding coccidioidal focus, fresh cavitation later appeared in the remaining right lower lobe.

In the differential diagnosis of the pulmonary fungus infections, a history of residence in an area endemic for such infections as histoplasmosis or coccidioidomycosis, or of an occupational exposure such as chicken-raising in the case of aspergillosis, are helpful clues. Cavitation, with sputum negative for tubercle bacilli and in the presence of a negative tuberculin skin test should prompt a search for fungus in the sputum, and histoplasmosis may be diagnosed by the presence of the organism in the bone marrow even when it is absent from the sputum. Skin-tests for the various fungi are suggestive, though not diagnostic, while complement fixation tests are more specific.

The uncertainty of the diagnosis when sputum is negative is demonstrated by cases 3 and 4. Although we felt that we had ruled out tuberculosis before surgery, the infecting organism was not isolated until the pathological specimen was examined after operation.

Case 3: W.O., a 30 year old white male, was admitted to the hospital February 7, 1950 with a history of cough productive of foul sputum since May 1949. He had suffered a 27 pound weight loss. A chest x-ray film had

FIGURE 3 FIGURE 4

Figure 3, Case 3, W.O.: Tomogram showing large cavity. Nocardia.
Figure 4, Case 4, O.H.: Cavity right upper lobe. Aspergillosis.

been interpreted as showing virus pneumonia, for which he was treated with penicillin. The sputum temporarily decreased, but he then developed chest pain and fever up to 102 degrees F. He was admitted to another hospital where pus was aspirated from his right chest and he was treated with penicillin, streptomycin, and chloromycetin. He was improved for two or three months, but about six weeks prior to admission again developed cough productive of foul sputum, weight loss and fatigue. Physical examination on admission showed a chronically ill man with clubbing of the fingers, flattening of the right thorax, with decreased respiratory excursions on that side, dullness to percussion and diminished breath sounds.

X-ray and laboratory findings: Posterio-anterior chest x-ray film and tomograms showed large cavities in the apex of the right lower lobe and in the right middle lobe. WBC 21,300 with 82 per cent polymorphonuclears. Repeated sputum examinations were negative for tubercle bacilli.

Right pneumonectomy was performed on February 22, 1950. The resected lung showed involvement of all three lobes in a granulomatous process with multiple cavitation. Microscopic examination and smear of material from the cavities showed gram positive, non-acid fast filaments exhibiting true branching, which were identified as that group of actinomycosis known as nocardia. The final diagnosis was actinomycosis, right lung.

Case 4: O.H., a 53 year old white male, was admitted to the hospital on September 2, 1950 with a chief complaint of cough and right chest pain since August 1950. His cough was productive of thick flocculent yellow and gray sputum, and an x-ray film taken by a private physician was interpreted as showing pulmonary tuberculosis. This diagnosis was confirmed by a second physician after another x-ray film inspection, and the patient was referred to us with a diagnosis of active pulmonary tuberculosis with cavitation. One sputum test before admission was negative for acid fast bacilli.

Physical examination on admission showed a poorly nourished man with clubbing of the fingers and toes. There were increased tactile and vocal fremitus and bronchial breath sounds over the upper third of the right lung anteriorly.

X-ray and laboratory findings: A chest x-ray film on admission showed marked upward retraction of the right pulmonary artery with a large cavity at the right apex and another cavity at the left apex. Bronchoscopy was negative. Nine sputum tests and bronchial aspirations were negative for acid fast bacilli; Papanicolau smear of two sputum specimens and of bronchial aspirations were negative for malignant cells.

A pre-operative diagnosis of infected emphysematous blebs was made, and right upper lobectomy was performed on October 25, 1950. At operation the right upper lobe was found to be densely adherent to the chest wall because of thick fibrous adhesions. The excised lobe felt solid, and on section showed a large, thick-walled cavity containing dark, fecal-appearing but odorless material. Microscopic study and smear of the material and of the cavity wall yielded organisms characteristic of aspergillus. No other organisms were found. After a stormy post-operative course he made a complete recovery and was symptom-free on discharge. The final diagnosis was pulmonary aspergillosis, although it cannot be definitely stated whether aspergillum is ever a primary cause of lung pathology or merely a secondary invader of pre-existing lesions.

Cystic Disease of the Lung: The pathogenesis of cystic disease of the lung is still a matter of debate. It is no longer universally accepted to be due to congenital malformation of the lung, though Theodos[14] feels that there have been enough well-authenticated cases of cystic disease apparently present from birth to prove that the condition may be congenital. Cunningham and Parkinson[15] made a detailed histological study of six cases in which they concluded that the common pathogenesis was a non-specific, non-infectious granulomatous process. It is debatable whether infection can produce cystic changes in the lung or whether the infection is imposed secondarily upon already present cystic changes. Wherever multiple small cysts are present, the x-ray picture must be differentiated from other cavernous pulmonary disease. In case 5, the diagnosis was complicated by a strong family history of tuberculosis, so that despite the persistent absense of tubercle bacilli from the patient's sputum and a remarkably benign course in the presence of multiple cavitation, she was treated for more than a year for tuberculosis.

Case 5: V.K., a 19 year old white female, was admitted on September 3, 1949 with a referring diagnosis of pulmonary tuberculosis, far advanced. The patient's father had been hospitalized for tuberculosis when she was eight years old; at that time her tuberculin skin test was positive, but her x-ray film was negative. Follow-up films were taken, and in 1947 her x-ray film was interpreted as being "suspicious of tuberculosis"; a review of previous films at this time was said to show that x-ray films since 1943 had been suggestive of tuberculosis with cavitation. In July 1948 she was admitted to a sanitorium in Iowa, and a report of her admission film to that institution read: "caseous pneumonic tuberculosis, far-advanced, with a cavity in the right apex and an infiltrative

FIGURE 5a FIGURE 5b

Figure 5a, Case 5, V.K.: April 21, 1950, before resection.—*Figure 5b:* Same patient, August 18, 1950, three months after resection with new annular shadows right mid-lung field.

lesion in the right perihilar zone." However, despite this extensive disease, all sputa were consistently negative for tubercle bacilli, by smear, culture, and gastric lavage.

Physical examination showed slight dullness over the right lower lobe with increased tactile and vocal fremitus in this area. X-ray and laboratory findings: X-ray film inspection showed evidence of cavitation in the right upper, middle, and lower lobes, and by April 27, 1950 the cavity in the right lower lobe had increased in size and showed a fluid level. Tuberculin skin test was positive 1:10,000. Intradermal histoplasmin and coccidiodin tests were negative. Bronchoscopy was negative. Twenty sputum concentrates, two bronchial lavage concentrates, and seven sputum cultures were all negative for acid fast bacilli; three sputa were negative for fungi.

Because of the increasing size of the cavity, an exploratory thoracotomy was done on May 24, 1950. The entire upper lobe and the superior segment of the lower lobe were involved in a granulomatous process. The right upper lobe and apical segment of the lower lobe were resected. Smears of the tissue, and cultures of it for acid fast bacilli were all negative, as were aerobic and anairobic culture for other organisms. Microscopic examination of the tissue presented a picture in no way suggestive of tuberculosis or fungus infection; the sections were shown to several pathologists who agreed that it revealed a non-specific granulation tissue.

The patient recovered uneventfully from surgery, but began to raise blood-streaked sputum. An x-ray film taken on August 18, 1950 showed the development of new, thin-walled annular shadows on the right side. There was also an annular shadow on the left, and therefore further resectional surgery was not considered. When hemoptysis subsided she was discharged home in good clinical condition. Final diagnosis: Cystic disease of the lung with secondary infection.

Neoplasm: According to Reisner[16] cavitation in pulmonary neoplasm may be due to aseptic necrosis in rapidly expanding tumor tissue with a deficient blood supply, secondary infection due to

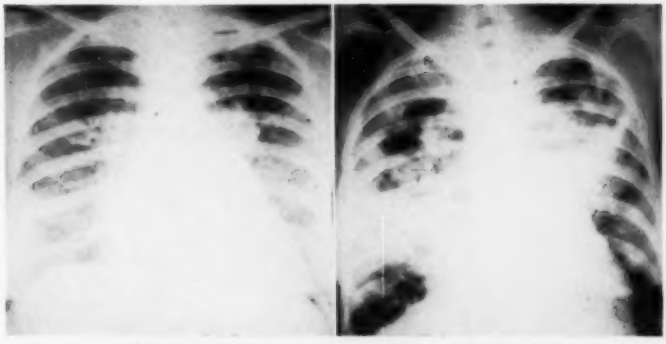

FIGURE 6a FIGURE 6b

Figure 6a, Case 6, M.J.: May 6, 1950. Rheumatic heart disease, no cavitation. *Figure 6b:* Same patient, June 1, 1950, massive pulmonary infarction with bilateral cavities.

blockage of communicating bronchi with retention of secretions, or hemorrhagic infarction following thrombosis of pulmonary vessels in the region of the tumor. Although, in general, the cavities seen in carcinoma of the lung tend to be more shaggy and irregular in outline than tuberculous cavities, it is usually impossible to make a definitive differential diagnosis on the basis of the roentgen picture alone.

So much has been written on the diagnosis of pulmonary neoplasm, that we shall merely summarize briefly here. Examination of the sputum, bronchial washings, and pleural fluid for malignant cells, bronchoscopy with biopsy, lung biopsy, and finally exploratory thoracotomy, may have to be done for a persistent pulmonary lesion in which tuberculosis or other specific etiology cannot be found.

Pulmonary Infarction: Cavitation may occur following pulmonary infarction, whether that infarction is caused by emboli occluding the pulmonary vessels, or local changes in the vessels in thrombosis or in polyarteritis. Sweeney and Bagenstoss,[17] and Sandler, Matthews and Bornstein[18] have recently described patients with multiple pulmonary excavations proved at autopsy to be due to infarction secondary to local vascular changes of polyarteritis.

Three different mechanisms operate in the production of cavitation by pulmonary emboli. Septic infarcts may produce localized lung abscesses, similar abscesses may occur following sterile emboli infected after they reach the lung, as reported by Levin and his group,[19] or massive embolization may produce sterile necrosis of lung tissue, with excavation, in the absence of any primary or secondary infection.

FIGURE 7

FIGURE 8

Figure 7, Case 7, L.C.I.: Giant cavity right upper lobe due to septic pulmonary infarction.—*Figure 8, Case 8, E.S.:* Traumatic cavity left mid-lung field.

Case 6 is an example of massive sterile necrosis of lung tissue, while case 7 illustrates the production of lung abscess by emboli from an infected source.

Case 6: M.J., a 35 year old white female, was first seen in the emergency room on May 5, 1950 because of substernal pain and hemoptysis. The patient had a history of rheumatic heart disease since the age of nine, with frequent bouts of congestive failure since she was 18. Chest x-ray film on May 5th showed only a markedly enlarged heart with mitralization of the left border. She was transferred to a private hospital, from which she was sent back again on June 3, 1950. At this time she was acutely ill with a temperature of 102 degrees F. and severe dyspnea even while receiving oxygen. There was a thrombophlebitis of the left arm. Coarse rhonchi were heard throughout both lung fields, with bronchial breath sounds in the upper third of both lungs. The cardiac rhythm was totally irregular at a rate of 74; an apical diastolic thrill and apical diastolic and systolic murmurs were present.

Laboratory and x-ray findings: X-ray film on June 3rd showed multiple bilateral cavitations, with pleural effusion on the right, and multiple pulmonary infiltrates. Urinalysis was negative for albumin, sugar, and acetone but showed many pus cells. WBC 36,400 with 95 per cent neutrophiles.

The patient's course was downhill; on the afternoon of June 4th her respiration became rapid, she began to bring up frothy, bloody sputum, and died shortly afterward. Autopsy showed large excavated infarcts in both lungs, with adherent clot and partially organized thrombi in the supplying arteries. Smear and culture made from material in the cavities were negative for acid fast bacilli, fungi, and other organisms, indicating that this was a true sterile necrosis of lung tissue.

Case 7: L.C.I., a 39 year old white male, was transferred from another hospital on July 28, 1950 with a diagnosis of pulmonary tuberculosis. He gave a history of occasional chilly and feverish feelings since January 1950, with increased cough and sputum since June 1950, and blood-streaked sputum with right chest pain, since July. On July 18th he had had a pulmonary hemorrhage of about 90 cc. Further questioning elicited a history of pain and swelling in the left leg since the beginning of July, which had increased to the point where he was unable to walk.

Physical examination showed evidence of recent weight loss. There was some impairment of the percussion note in the right axilla. Varicose veins were present in the left leg.

X-ray and laboratory findings: An x-ray film on July 31, 1950 showed a rather homogeneous parenchymal infiltration of the right upper lobe, with two thick-walled cavities each measuring approximately 1.5 cm. in diameter. By August 12, 1950 an x-ray film showed multiple areas of cavitation in the right upper lobe, one of them measuring 5 cm. in diameter. Three smears and 10 cultures were negative for tubercle bacilli; cultures were negative for Friedlander's bacillus, but grew a pure culture of E. coli.

The patient was treated with aureomycin with good clinical results and with rapid progressive clearing of the pulmonary lesions by x-ray film. A ligation and stripping of the left greater saphenous vein was done on January 2, 1951. Final diagnosis: Abscess of lung with cavitation secondary to pulmonary embolism from infected left saphenous varicose veins.

Trauma: Occasionally, chest x-ray films taken subsequent to a non-penetrating chest injury reveals cavitation which is interpreted as indicating a hitherto undiagnosed pulmonary tuberculosis. However, review of the experimental work of Moolten[5] mentioned above, shows that purely mechanical cavities can be produced by rupture of lung tissue with resultant retraction of elastic tissue. Serial x-ray films on patients with cavitation secondary to trauma, will show rapid healing of the cavities, with negative laboratory findings throughout.

Case 8 is an instance in which cavitation appeared following non-penetrating trauma to the chest wall, and on whom an initial diagnosis of pre-existing tuberculosis was made.

Case 8: E.S., a 29 year old white male, was admitted to the hospital following a car accident in which he sustained a blunt crushing injury to the left chest. There was fracture of the left third and fourth ribs, with a traumatic pneumothorax on that side. An x-ray film taken aftel aspiration of the pneumothorax showed a cavity in the left upper lobe, and a provisional diagnosis of pulmonary tuberculosis was made. Tuberculin skin test was positive, but 10 sputa were negative for acid fast bacilli and the patient denied any previous history of tuberculosis. An x-ray film taken 10 days after the accident showed the cavity entirely gone, and therefore a final diagnosis of traumatic cavity was made.

Note: Cases 1, 2, and 7 are included by courtesy of Fitzsimmons General Hospital. Case 6 by courtesy of Colorado General Hospital. Case 8 by courtesy of Denver Gentral Hospital.

SUMMARY

In considering the numerous pathological conditions of the lung which can produce cavitation and mimic tuberculosis, we do not overlook the many instances in which cavernous tuberculosis may be present with sputum persistently negative, even over periods of many years.

However, before making the diagnosis of tuberculosis in a patient with negative sputum whose x-ray picture may be indistinguishable from that produced by tuberculosis, exhaustive studies must be done to rule out other etiological agents. Papanicalaou smears for malignant cells, fungus studies of the sputum plus skin testing and serologic tests for the cavity producing fungus diseases, cardiac evaluation and studies of other organ systems for andications of polyarteritis, should all be part of the work-up of the patient with pulmonary cavitation and negative sputum. Even then, in some instances, final diagnosis cannot be made except by the pathologist, but many mistaken diagnoses of tuberculosis and tragic delays in surgery, will be avoided.

RESUMEN

Considerando las numerosas condiciones patológicas del pulmón que pueden producir la excavación y simular tuberculosis, no pasamos por alto los muchos casos en que puede existir la tuberculosis excavada, con esputos persistentemente negativos, aún durante períodos de muchos años.

Sin embargo, antes de dar un diagnóstico de tuberculosis en un paciente con esputo negativo, cuyas radiografías puedan no distinguirse de las de tuberculosis, es preciso llevar a cabo estudios minuciosos con el fin de excluir otros agentes etiológicos. Frotis de Papanicolau en busca de células malignas, estudios del esputo buscando hongos, además de las pruebas cutáneas y serológicas con relación a las enfermedades micósicas que producen excavación, la función cardíaca y estudios de otros sistemas orgánicos buscando indicaciones de poliarteritis—todo esto debe incluirse en la exploración del paciente que tiene excavación pulmonar y esputo negativo. Aún haciéndose todo esto, en algunos casos el diagnóstico final sólo lo podrá dar el patólogo, pero se evitarán muchos diagnósticos equivocados de tuberculosis y retrasos trágicos de la cirugía.

RESUME

En considération du nombre important d'affections pulmonaires qui peuvent s'accompagner de cavernes et simuler la tuberculose, les auteurs attachent une attention particulière sur les cavités tuberculeuses qui peuvent exister malgré une expectoration constamment négative, même pendant plusieurs années.

Toutefois, il est nécessaire de faire une étude complète du malade, pour éliminer toute autre étiologie quand on se trouve en présence de tels faits. Il faut pratiquer l'épreuve de Papanicolaou pour rechercher les cellules cancéreuses; la recherche des mycoses par l'examen des crachats, et par les tests cutanés et sérologiques; l'étude de l'état cardiaque et l'examen general pour rechercher les manifestations athéromateuses.

Malgré tout, dans quelques cas particuliers, le diagnostic véritable ne peut être fait que par l'anatomie-pathologique. Cependant, on peut éviter ainsi de nombreux diagnostics erronnés de tuberculose, et de tragiques atermoiements dans l'intervention chirurgicale.

REFERENCES

1 Corylios, P. N.: "The Mechanics and Biology of Tuberculous Cavities," *Am. Rev. Tuberc.*, 33:369, 1936.
2 Andrus, P. M.: "The Pathogenesis of Tuberculous Cavities," *Am. Rev. Tuberc.*, 38:174, 1938.
3 Andrus, P. M.: "Bronchiectasis: An Analysis of Its Causes," *Am. Rev. Tuberc.*, 36:46, 1937.

4 Reinders, D.: "Uber Form und Grosse der Fruhkaverne," *Ztschr. f. Tuberk.*, 51:438, 1928.
5 Moolten, S. E.: "Mechanical Production of Cavities in Isolated Lungs," *Arch. Path.*, 19:825, 1935.
6 Cohen, S.: "Resolving Lobar Pneumonia in Adults Simulating Tuberculosis in the Roentgenogram," *Am. J. Roentgenol.*, 40:202, 1938.
7 Sellors, T. H., Blair, L. G., Houghton, L. E., Thompson, V. C. and Pryce, D. M.: "Spreading Suppurative Pneumonitis," *Thorax*, 1:146, 1946.
8 Hobby, A. W.: "Pulmonary Mycoses," *Dis. of Chest*, 15:174, 1949.
9 Furcolow, M. L.: "Further Observations on Histoplasmosis: Mycology and Bacteriology," *Public Health Report*, 65:965, 1950.
10 Chortis, P. A.: "A Case of Aspergillosis of the Lung," *Dis. of Chest*, 15:222, 1949.
11 Smith, C. E., Beard, R. R. and Saito, M. T.: "Pathogenesis of Coccidioidomycosis with Special Reference to Pulmonary Cavitation," *Ann. Int. Med.*, 29:623, 1940.
12 Berke, R., Schomer, A. and Bass, H. E.: "Residual Cavities in Pulmonary Coccidioidomycosis: Follow-up Studies," *Dis. of Chest*, 17:84, 1950.
13 Krapin, D. and Lovelock, F. J.: "Recurrence of Coccidioidal Cavities Following Lobectomy for a Bleeding Focus," *Am. Rev. Tuberc.*, 58: 282, 1948.
14 Theodos, P. A.: "Cystic Disease of the Lung with Report of a Case," *Dis. of Chest*, 14:115, 1948.
15 Cunningham, G. J. and Parkinson, T.: "Diffuse Cystic Lungs of Granulomatous Origin," *Thorax*, 5:43, 1950.
16 Reisner, D.: "Carcinomatous Cavities of the Lungs," *Quart. Bull., Sea View Hosp.*, 1:322, 1936.
17 Sweeney, A. R. and Baggenstoss, A. H.: "Pulmonary Lesions of Periarteritis Nodosa," *Proc. Staff Meet., Mayo Clinic*, 24:35, 1949.
18 Sandler, B. P., Matthew, J. H. and Bornstein, S.: "Pulmonary Cavitation Due to Polyarteritis," *J.A.M.A.*, 144:754, 1950.
19 Levin, L., Kernohan, J. W. and Moersch, H. J.: "Pulmonary Abscess Secondary to Bland Pulmonary Infarction," *Dis. of Chest*, 14:218, 1948.

Chronic Infectious Pulmonary Changes Simulating Tuberculosis in the Older Age Group

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Introduction

There is statistical proof of an increasing incidence and mortality from tuberculosis in individuals (particularly males) over 45 years. The mortality rate in this group in the United States has more than doubled since the beginning of the century (from 24 per 100,000 in 1900 to 52 in 1947).[1] While the lengthening of the life span has thus projected the importance of the tuberculosis problem, the physician will also commonly encounter cases with clinical pulmonary features, in the older age group, which may simulate tuberculosis. Pulmonary changes may result from repeated infectious insults (non-tuberculous) over a period of many years. Reference is made specifically to the pathological changes of extensive fibrosis, emphysema and chronic organizing pneumonitis, which may be quite diffuse, and which may be seen, for example, as late complications of bronchiectasis. The roentgenological findings, particularly in these patients, may be confused at times with that of tuberculosis, especially an old "burnt out" infection. The differential diagnostic problems and therapeutic implications, thus, assume importance. To emphasize and focus attention on this aspect of geriatrics, the following report briefly presents the clinical, radiological and pathological findings in three patients who were admitted to the hospital initially with the diagnosis of tuberculosis. All of them revealed extensive and disabling pulmonary changes of a non-specific character.

Case Reports

Case 1, Clinical Data: I.D., was admitted to this hospital for the first time on December 4, 1944 at the age of 75 years. There was a positive family history for tuberculosis. She had a chronic cough with non-foul expectoration (one to three ounces daily), for about 50 years, with attacks of pneumonia in 1903, 1911, and 1940. Dyspnoea had been noted for a number of years, together with episodes of fever and fatiguability. An hemoptysis on December 1, 1944 precipitated her entrance into the hospital. Coarse rales were heard throughout the greater portions of both lung fields. The roentgenogram of the chest revealed an increased density in the right hilar region, with scattered linear and nodular infiltra-

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tion, more concentrated in the lower half of the right lung field; there was some hyper-aeration of both lungs and flattening of the right diaphragm. The heart, while not enlarged, showed obliteration of the normal convex contour indicative of adhesions between the mediastinal pleura and parietal pericardium. The patient's clinical course was essentially uneventful. Thirteen sputum smears and nine culture examinations were negative for tubercle bacilli. She was discharged on February 17, 1945 with the diagnosis of pulmonary fibrosis, emphysema, bronchiectasis (bronchogram not performed).

Re-admission occurred on May 2, 1947 because of aggravation of cough, expectoration, dyspnoea and the development of nausea, vomiting, epigastric pain and dependent edema of several weeks duration. In addition to the signs of senility and abnormal pulmonary findings, there were signs of cardiac insufficiency, which improved with cardio-therapy. The chest x-ray film of May 5, 1947 (Figure 1) showed no significant change compared to the previously described film of December, 1944. Laboratory examination revealed: negative sputum for tubercle bacilli, anemia and leukocytosis, four plus albuminuria with granular casts and occasional red blood cells, and a rise in the blood urea from 39.5 mg. on admission, to 66.5 mg. on May 14. Death occurred on May 28, 1947.

Autopsy: Gross examination: (a) Bilateral pleural fibrous adhesions were present. The markedly emphysematous lungs showed apical scars which on section involved about 0.5 cm. of the parenchyma, with scattered dilated bronchi adjacent to the scar tissue. In the right upper and middle lobes and apical portion of the lower, there were a number of firm areas of scar tissue with organization of the lung in regions not exceeding 1.0 cm. in width, and about 1.5 cm. in length. At these sites, a number of dilated bronchi were filled with thick purulent exudate. Scattered in both lungs was evidence of hemorrhagic edema fluid. There was no evidence of tuberculosis; (b) Dilatation of both ventricules and slight hypertrophy of the left ventricles were noted together with ad-

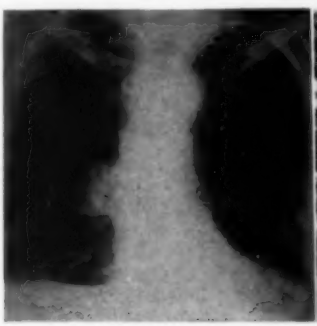

FIGURE 1

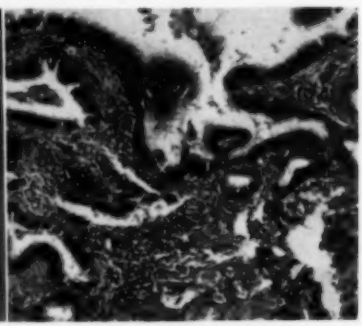

FIGURE 2

Figure 1, Case 1: X-ray May 5, 1947. Increased density right hilar area with scattered linear and nodular infiltration, more marked in right lower lung field; hyper-aeration of both lungs with flattened right diaphragm. Cardiac contour straightened. — *Figure 2, Case 1:* Lung, right upper lobe, medium power, extensive bronchiolar dilatation with surrounding inflammatory reaction and organization of parenchyma.

vanced atheromatous changes and areas of calcification in the aorta and larger vessels; (c) Extensive amyloid infiltration of both kidneys (contracted) and spleen, and to a lesser extent of the liver were evident.

Microscopic examination: Sections of both upper lobes showed wide areas of fibrosis with marked dilatation of the bronchi and proliferation of the bronchial epithelium. There was extensive inflammatory cell infiltration with large collections of lymphocytes, and in many areas, marked increase in connective tissue, with complete obliteration of the parenchyma inbetween (Figure 2 shows section in right upper lobe). Throughout the remainder of the right and left lung, there were areas of bronchial dilatation with cellular infiltration of the walls. Elsewhere there was merely dilatation of the alveoli and mild congestion.

Case 2, Clinical Data: M.K., a white female, age 67, was admitted March 31, 1947. She developed chronic cough and expectoration following an attack of influenza in 1918. Dyspnoea had been present on moderate physical exertion for several years. A loss of 20 pounds took place within a year. Following an x-ray examination of the chest, hospitalization was advised. On physical examination, the patient was thin and not dyspnoeic at rest. Dullness and rales were elicited over the greater portion of both lungs. The roentgenogram on admission revealed disseminated bilateral fibrosis, more marked on the right. There were many areas of "highlight" strongly suggestive of emphysema and bronchiectasis; irregularity of both diaphragms, and calcification of the tracheal rings and some of the larger bronchi, were noted. The cardiac silhouette was distorted, and the aorta widened. An electrocardiogram was within normal limits. All sputa examinations (15 smears and nine cultures) were negative for tubercle bacilli. Her symptoms improved, and she was discharged July 31, 1947. The diagnosis was pulmonary fibrosis, emphysema, and bronchiectasis.

She felt fairly well until weakness forced her to become bedridden in August, 1948. The occurrence of hemoptyses in February and May, 1949

FIGURE 3

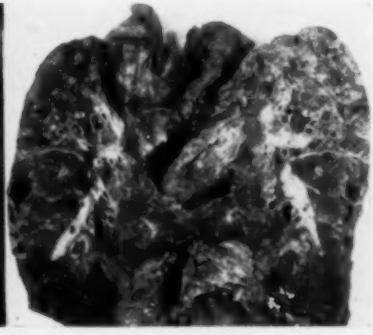

FIGURE 4

Figure 3, Case 2: X-ray (portable), May 10, 1949. Note multiple areas of "highlite" and fibrosis, especially in right upper lung field; both diaphragms irregular. Heart shadow distorted and aorta widened.—*Figure 4, Case 2:* Section of right lung revealed cystic bronchial dilatation particularly of upper and middle lobes with areas of organization.

FIGURE 5 FIGURE 6 FIGURE 8

Figure 5, Case 2: Lung, left upper lobe, medium power, area of organizing pulmonary tissue with lymphocytic infiltration of alveolar septa. Note the extensive fibrosis.—*Figure 6, Case 3:* X-ray February 26, 1949. Note multiple areas of radio-lucency, particularly in dense RUL; scattered linear infiltration elsewhere with some flattening of diaphragm. Loss of normal convex cardiac contour present.—*Figure 8, Case 3:* Lung, right lower lobe, medium power, dilated bronchi in an area of organized pulmonary tissue with large collection of lymphocytes. Note the sharp division from the adjacent dilated alveoli.

accounted for her re-admission on May 10, 1949. The chest x-ray film (portable), of that date (Figure 3), was essentially the same as that taken in 1947. The urinalysis showed four plus albuminuria, but the blood urea and creatinine were normal, as was the blood pressure. The patient died May 18, 1949.

Autopsy: Gross examination: (a) Both lungs had a somewhat similar appearance—firm and almost airless in the upper portions. The right upper lobe (Figure 4) had a honey-combed appearance due to diffuse dilatation of the branch bronchi, and interspersed, was organization of the parenchyma and anthracotic deposits. The middle lobe and apical portion of the lower lobe (Figure 4) showed similar cystic dilatation, with surrounding small areas of organized pulmonary tissue. The base of the lower lobe showed a rather normal architecture. The lingular portion of the left upper lobe was diffusely cystic also, with areas of organized tissue. The trachea and larger bronchi were mildly congested. There was no evidence of tuberculosis. The tracheobronchial lymph nodes were enlarged and firm; (b) Both ventricles were moderately dilated and atherosclerotic changes of the larger systemic vessels were apparent; (c) The kidneys, liver and spleen revealed moderate amyloid infiltration.

Microscopic examination: For the most part, multiple sections of the right lung showed the following: There were many cystically dilated bronchi with vascularization of the walls and almost complete destruction of the muscle coats. The peribronchial vessels were partially obliterated by pronounced intimal proliferation. The mucosa was ulcerated and there was diffuse inflammatory reaction. The surrounding tissue showed extensive connective tissue organization, focal collections of lymphocytes and anthracotic pigment, and small areas of residual alveoli lined by cuboidal epithelium. In areas beneath the pleura, there were

FIGURE 7, Case 3: Section of right lung showed shrunken upper lobe with overlying thickened pleura, dilatation of branch bronchi and parenchymal organization.

scattered dilated alveoli lined by cuboidal epithelium, and filled with an exudate of polymorphonuclear leukocytes and pink staining material. The entire inflammatory process was non-specific in character. Multiple sections of the left lung showed a process comparable to that seen on the right. A section (Figure 5) of the left upper lobe revealed an area of organizing pulmonary tissue with lymphocytic infiltration of alveolar septa and extensive fibrosis.

Case 3, Clinical Data: M.F., a white female, age 73, was transferred to this hospital on December 3, 1946. She was supposed to have had rheumatic fever at the age of 16 years. For almost 25 years, she had episodes of exertional dyspnoea, orthopnoea, palpitation, chronic cough and expectoration. During this time she was "labelled" as a "cardiac," and led the life of a semi-invalid. For the past four years she had noted increasing frequency of precordial pain. Early in November, 1946, she developed chilliness, fever, fatiguability, and was admitted to the Jersey City Medical Center on November 28th. Because of the appearance of the chest x-ray film, and a single positive sputum report for tubercle bacilli, she was subsequently transferred. Physical examination disclosed a poorly nourished, somewhat dyspnoeic female. Dullness was noted over the right side posteriorly, and the left upper lung field anteriorly, with coarse rales in the same areas, and bronchial breathing at the right apex. A systolic murmur was heard at the apex, but the rhythm was regular and the blood pressure normal. The electrocardiogram revealed left axis deviation. Sputa examinations (13 smears and 11 cultures) were negative for tubercle bacilli. The roentgenogram of November 27, 1946 showed slight displacement of the trachea to the right. Within the dense shrunken right upper lung field, demarcated by the thickened horizontal fissure there were a number of areas of increased radiolucency; infiltration of a similar type, but less confluent, were noted also in the lower third of the right lung, and scattered throughout on the left. Both diaphragms were somewhat irregular, and loss of the normal convex cardiac contour was apparent. The patient was discharged on May 31, 1947 with the final diagnosis of pulmonary emphysema, fibrosis, and bronchiectasis.

The symptoms of vomiting and diarrhea led to her readmission to the Medical Center on February 26, 1949. Her gastric symptoms subsided shortly afterward, but because of the chest x-ray findings, she was again transferred to the Pollak Hospital on March 4, 1949. Cough and expectoration (non-foul and non-bloody), were moderate. Physical and x-ray film examination of the chest (Figure 6) were essentially the same as on the previous admission. Twelve sputum smears and seven culture studies were negative for tubercle bacilli. Urinalysis and blood urea were normal. On May 25, 1949 she suddenly developed convulsions, and died soon thereafter.

Autopsy: Gross examination: (a) The right lung (Figure 7) showed considerable thickening and hyalinization of the pleura over the upper lobe, which was reduced in volume. On section, there was a cystic appearance due to marked dilatation of branch bronchi, with a condensation of the bronchopulmonary structures, and diffuse organization of the intervening parenchyma. The lateral portion of the upper lobe was most involved. In a similar manner, the lateral portion of the lower lobe beginning at the apex and extending to the base, showed cystic dilatation of the branch bronchi, surrounded by organized pulmonary tissue. The axillary portion of the middle lobe was partially organized, and the

bronchi dilated; broad linear scars were also seen. All of these changes were non-specific.

The changes in the left lung were less marked. Again, cystically dilated branch bronchi surrounded by organized tissue was seen in the apex of the upper lobe, the lingula, and upper part of the lower lobe, with interspersed linear scars. The remainder showed fairly normal architecture.

The tracheobronchial lymph nodes were enlarged, anthracotic, and contained calcific foci: (b) There was hypertrophy of the right ventricle, dilatation of both ventricles, with focal myofibrosis of the posterior wall of the left ventricle.

Microscopic examination: The right and left lungs showed almost identical findings. Sections through the upper lobes revealed marked bronchial dilatation, considerable vascularity of the bronchial walls, diffuse inflammatory cell infiltration, and dense peribronchial connective tissue. Some areas showed marked dilatation of alveoli adjacent to areas of organized pulmonary tissue, within which were scattered deposits of anthracotic pigment and /or collections of lymphocytes. Some of the alveoli and bronchi were filled with an exudate of leukocytes. The lower lobes presented a somewhat similar picture. Figure 8, taken from a section through the right lower lobe, showed dilated bronchi in an area of organized pulmonary tissue, with a large collection of lymphocytes. (Note the sharp division from the adjacent dilated alveoli).

In the left upper lobe, in addition to the above findings, there were a number of small areas in which the centers were caseous, with a peripheral zone of connective tissue marking off the caseous foci. These lesions were discrete, and there was no evidence of any reactivation in the old tuberculous foci.

Comment

In treating elderly patients, the physician not infrequently is confronted with clinical findings which are the end result of many factors. It therefore becomes important to evaluate their medical problems both from the basic standpoint of etiology and resultant conditions. It is recognized that in the *normal* process of aging, certain structural and functional changes affect the thoracic cage and lungs; in the latter there occur diminished elasticity of the parenchyma, some increase in interstitial connective tissue, and variable impairment of the ciliary and broncho-motor functions. This defect will favor aspiration and retention of secretions in older people, and predispose to bronchitis, atelectasis and pneumonia. The effects of chronic antecedent pulmonary infection may become superimposed on this local aging process. To be content simply with a diagnosis of chronic bronchitis in the aged, can sometimes have disastrous consequences. Chronic bronchitis in this group is usually secondary to some more important and specific condition, such as cardiac insufficiency, bronchial asthma, carcinoma, tuberculosis, bronchiectasis.

In this report, the late anatomical changes of long standing pulmonary (non-specific) infection are emphasized. The three

cases exhibited essentially similar findings, consisting of diffuse emphysema, fibrosis, and areas of chronic inflammation—secondary undoubtedly to the bronchiectasis. A knowledge of the development of such late irreversible sequelae, may stimulate consideration of resectional therapy for bronchiectasis when feasible earlier in life. It is interesting to note also, that two of the patients showed amyloid changes secondary to the chronic pulmonary infection.

These cases were admitted with the diagnosis of pulmonary tuberculosis. Cough and expectoration of many years' duration was an important clinical clue to the correct diagnosis, although the roentgenologic findings, by themselves, particularly in Cases 2 and 3, simulated tuberculosis. Cases 1 and 2 developed hemoptyses resulting from bronchiectasis. Sputum examinations are of paramount importance, of course, in considering the diagnosis of tuberculosis. Unnecessary isolation, protracted hospitalization, and mistreatment can be avoided, when tuberculosis is ruled out. In Cases 1 and 2, many sputum studies were negative for tubercle bacilli by concentrated smear and culture, and the autopsy in each instance, revealed no evidence of tuberculosis. The third patient was diagnosed as a "cardiac" for 25 years, but she was actually a "pulmonary cripple." A single positive sputum was obtained prior to her transfer to the chest hospital, where this report was not confirmed, although the post-mortem examination revealed some microscopic encapsulated tuberculous foci, which actually constituted an incidental finding. The remainder of the pulmonary findings was non-specific and unrelated to tuberculosis.

Appreciation is expressed to Dr. I. E. Gerber, Director of the Laboratory of the Pollak Hospital for Chest Diseases, for his contribution in the preparation of this manuscript.

SUMMARY

Bronchiectasis, particularly when untreated, may result after a period of time in the development of rather widespread anatomical changes such as emphysema, fibrosis, chronic pneumonitis, adhesive pleurisy. Cystically dilated bronchi and emphysematous areas may be mistaken for pulmonary cavitation on the roentgenogram of the chest and lead to a diagnosis of tuberculosis, especially when these shadows appear in the upper lung fields.

Increasing medical attention is being placed today on the care of the chronically ill and the older age group in the population. Among the latter, pulmonary tuberculosis has shown an increased incidence. The late complications of bronchiectasis mentioned above, may simulate tuberculosis (particularly the old chronic fibroid type). This report presents the clinical, radiological and

pathological findings in three elderly patients who were diagnosed on admission as tuberculous. All of them revealed extensive pulmonary damage of a non-specific character secondary to bronchiectasis. The differential diagnostic problem is a frequent and important one, but not always an easy one. The clinical history and the search for tubercle bacilli are significant aids. The availability of an increasing spectrum of antibiotics to combat infection, and the use of broncho-dilator drugs for bronchospasm, may be helpful adjuvants in the symptomatic relief of these older individuals who have essentially irreversible pulmonary changes.

RESUMEN

La bronquiectasis, particularmente cuando no es tratada, puede, después de un lapso de tiempo, dar origen a cambios anatómicos bastante diversos, tales como el enfisema, la fibrosis, la neumonitis crónica, la pleuresía adhesiva. Los bronquios dilatados en forma quística y las áreas enfisematosas pueden ser confundidas con la excavación pulmonar en la radiografía del pecho y dar lugar a un diagnóstico de tuberculosis, especialmente cuando estas sombras aparecen en los campos superiores de los pulmones.

Hoy se presta más atención al cuidado de los enfermos crónicos y del grupo de mayor edad de una población. Entre éstos últimos, se ha manifestado un aumento en la incidencia de la tuberculosis pulmonar. Las complicaciones tardías de la bronquiectasis que antes se mencionan, pueden simular la tuberculosis (en particular el tipo antiguo crónico fibroso). Este informe presenta los datos clínicos, radiológicos y patológicos de tres enfermos entrados en años, a quienes se les diagnosticó tuberculosis al admitirlos. Todos acusaron un daño pulmonar extenso, de carácter no específico, secundario a la bronquiectasis. El problema de diagnóstico diferencial es frecuente e importante pero no siempre fácil. La historia clínica y la busca de bacilos tuberculosos son valiosas ayudas. La disponibilidad de una variedad cada vez mayor de antibióticos para combatir las infecciones y el uso de drogas bronquiodilatoras para espasmos bronquiales, pueden ser adyuvantes útiles para el alivio sintomático en estos individuos mayores que han sufrido cambios pulmonares esencialmente irreversibles.

RESUME

Les bronchiectasies, surtout lorsqu'elles ne sont pas traitées peuvent déterminer après un certain temps des altérations anatomiques, telles que l'emphysème, la fibrose, la pneumonie chronique, la symphyse pleurale. Les dilatations kystiques des bronches et les zones emphysémateuses peuvent être confondues avec une caverne pulmonaire à l'examen de la radiographie, et considérées

alors comme tuberculeuses, en particulier quand ces ombres siègent dans la partie supérieure du poumon.

Actuellement, le public médical s'intéresse de plus en plus au traitement des affections chroniques, et aux personnes âgées. Parmi cette dernière catégorie d'individus, la fréquence de la tuberculose pulmonaire s'est nettement accrue. Les complications tardives de dilatations des bronches qui ont été mentionnées ci-dessus peuvent simuler la tuberculose, en particulier la tuberculose fibreuse chronique. Ce rapport concerne les constatations cliniques, radiologiques, anatomo-pathologiques qui furent faites chez des malades âgés, qui avaient été admis à l'hôpital avec le diagnostic de tuberculose. Tous montrèrent des altérations pulmonaires étendues, de caractère non spécifique, et secondaire à la dilatation bronchique.

Le diagnostic différentiel est un problème qui se posefréquemment, qui est très important dans ces cas, mais que souvent n'est pas facile. L'histoire clinique et la recherche du bacille de Koch sont des éléments de valeur. La possibilité d'un accroissement de la gamme des antibiotiques, et l'utilisation de substances bronchodilatatrices peuvent être d'un grand secours dans l'amélioration de ces individus, chez lesquels existent essentiellement des altérations pulmonaires irréversibles.

REFERENCE

1 U. S. Public Health Reports, April 1, 1949.

Pneumoperitoneum with Late Results
Report of 560 Cases*

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Pneumoperitoneum has been used principally as a measure in treating tuberculous peritonitis and as an agent to prevent adhesions since 1893. However, not until 1931 was it given for any other purpose, and the first time was accidental, when in attempting to give pneumothorax to control a hemorrhage, pneumoperitoneum was the result. On this occasion the hemorrhage was stopped and pneumoperitoneum was then continued as the treatment for an extended period of time with hopeful results.

We first used this measure, in conjunction with phrenemphraxis, in 1937 and for the first two or three years it was only used on those cases for whom no real hope was entertained. They were all far advanced with bilateral involvement, positive sputum, and often bilateral cavitation. The prognosis was poor to hopeless in all of our early cases. We were so pleased with the results, even in this group, that we started in 1941 giving it to more hopeful cases and at present pneumoperitoneum is the orthodox form of temporary collapse treatment at our institution. In fact, for the past two years our fills of pneumoperitoneum have been 10 times as many as pneumothorax. We do not consider pneumoperitoneum a competitive treatment to pneumothorax, but in our opinion the indications for pneumothorax have narrowed so much in the past few years and the indications for pneumoperitoneum are still broad enough to make this our principal temporary collapse measure. We shall not, in this paper, attempt to give you the technique of this treatment, nor the indications, because that can be found in Banyai's Book on Pneumoperitoneum.[1]

In setting up our criteria for this paper, it was decided to adhere strictly to the National Tuberculosis Association's classifications and this we have done with the exception of that requirement referring to sputum tests, which is explained in another part of the paper. In addition to this, none of our cases are considered as successfully treated unless they have one of the classifications, apparently cured, etc., based on a report on these patients within the past year and this report included x-ray, degree of activity,

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whether or not pneumoperitoneum was discontinued and sputum report. In our cases that we considered prepared for thoracoplasty, our requirement was that, as a result of the pneumoperitoneum and phrenic nerve operation, these cases became suitable for this operative procedure and were at least fair surgical risks, and who in our opinion would not have been suitable for this without pneumoperitoneum. In setting a dead-line, we selected six months as the time necessary to give this type of collapse therapy a reasonable chance to be successful. Many of these cases, although we are classing them as failures, in our opinion definitely had their lives prolonged as a result of this treatment and were no discredit to the procedure.

Prior to 1945 we had given this treatment to 579 white men and women. It is our purpose at this time to give you the late results of pneumoperitoneum, usually in conjunction with temporary paralysis of one hemidiaphragm; 92½ per cent successful cases, 91 per cent unsuccessful cases; 94½ per cent unreported cases had temporary phrenic nerve operation in this group. Crow and Whelchel[2] reported the early results in this same group of cases in 1945 and our late study shows comparable results. In our early cases it was hard to decide which side was worse and very often it was a question of tossing a coin to decide the side on which the phrenic nerve should be crushed. Of this total, 119 took the treatment for less than six months and so are not considered in our study. This leaves 560 cases on which we will base our evaluation. These include 13 minimal, 210 moderately advanced and 337 far advanced cases of pulmonary tuberculosis. Of the moderately advanced group 112 had positive sputum and 98 were negative. Eighty-seven of the 112 have been converted, a percentage of 78+. Of the far advanced cases, 318 were positive, and 19 were reported as negative. One hundred sixty seven or 52+ per cent of these positive cases were converted. All of the minimal cases were negative.

TABLE I
NUMBER OF CASES AND SPUTUM RESULTS

	No.	Not Included	No. Cases Analyzed	Sputum Before Treatment		Conversion	
				Pos.	Neg.	No.	Pct.
Minimal	15	2	13	0	13	0	0
Moderately Advanced	240	30	210	112	98	87	78+
Far Advanced	424	87	337	318	19	167	52+
TOTAL	679	119	560	430	130	254	

We have personally been disappointed with the results on the minimal cases, because in a group of 13, we have two failures. In the minimal group one probably would get just as good results on bed rest. Of this 13, four are today apparently cured, one is arrested, and we have received no follow-up reports on six. One of these failures started treatment in February 1944 and in March 1946 the lesions were calcified. Pneumoperitoneum, however, was continued and in 1949 she developed peritonitis and died. The cause reported on the death certificate was tuberculous peritonitis. We have no report of the intervening years as to what happened to the pulmonary lesions, and we think it proper to place this patient in the failure group.

Of the 210 moderately advanced cases, 100 are apparently cured, 19 arrested, six quiescent and two were prepared for thoracoplasty. There were 44 failures and we have been unable to obtain follow-up reports on 39. Of these 210 cases only 12 are known to be dead and three of these were non-tuberculous.

TABLE II
RESULTS OF STUDY

	Apparently Cured	Arrested	Apparently Arrested	Quiescent	Prepared for Thoracoplasty	Failures	No Report	Known Deaths
Minimal	4	1	0	0	0	2	6	1
Moderately Advanced	100	19	0	6	2	44	39	12*
Far Advanced	52	31	1	4	26	178	45	92**
TOTAL	156	51	1	10	28	224	90	105

*3 deaths were non-tuberculous, M. A.
**3 deaths were non-tuberculous, F. A.

TABLE III
DISCHARGE STATUS

	Arrested	Apparently Arrested	Quiescent	Improved	Unimproved or Worse	Total
Minimal	2	0	1	2	1	6
Moderately Advanced	2	0	14	21	2	39
Far Advanced	0	1	6	33	5	45
TOTAL	4	1	21	56	8	90

Of the 337 far advanced cases, 52 are apparently cured, 31 arrested, one apparently arrested, four quiescent, 26 have been prepared for thoracoplasty, 178 have been failures and 45 have not been traced. In this group of far advanced cases practically all of whom had a questionable to hopeless prognosis, there are only 92 known deaths and three of these were due to non-tuberculous conditions, the pulmonary lesions at that time being quiescent. Of the total of 560 minimal, moderately advanced and far advanced cases of pulmonary tuberculosis, 246 or slightly less than 50 per cent, have been successfully treated with this type of collapse therapy. There is a total of 90 cases that we have been unable to get follow-up reports on but at discharge the classifications were: of the six minimal cases, two were arrested, one quiescent, two improved and one worse. Of the 39 moderately advanced cases, two were arrested, 14 quiescent, 21 improved and two unimproved or worse. Of the 45 far advanced cases, one apparently arrested, six quiescent, 33 improved and five unimproved or worse.

TABLE IV

LENGTH OF TIME PNEUMOPERITONEUM TAKEN: SUCCESSFUL CASES

	Minimal	Moderately Advanced	Far Advanced	Total
½- 2 years	1	28	15	44
2- 4 years	2	41	33	76
4- 5 years	1	18	15	34
5-10 years	1	40	49	90
TOTAL	5	127	112*	244

*2 F. A. No report on time.

TABLE V

LENGTH OF TIME PNEUMOPERITONEUM TAKEN: FAILURE CASES

	Minimal	Moderately Advanced	Far Advanced	Total
½- 2 years	0	13	72	85
2- 4 years	1	13	55	69
4- 5 years	0	5	17	22
5-10 years	1	10	27	38
TOTAL	2	41*	171**	214

*1 M. A. No report on time.
**9 F. A. No report on time.

In explanation of this rather large number of cases without follow-up, we believe the situation in Georgia may be somewhat different than in some other places. Our follow-up reports must be obtained from the 159 county health departments. So many of the reports would give us really beautiful statistics up to 1945, 1946 or 1947, but we have included in our group of successful cases none except those that had x-ray film inspection, sputum examinations and necessary time-up in the report, and this report within the past year.

We have also made an effort to come to some conclusions as to the time needed to continue this type of treatment. Of course, this varies in individual cases, but our statistical review shows the length of time that various patients have taken the treatment. The successful patients are listed in Table 4, which shows that of the five minimals, one took the treatment less than two years, two between two and four years, one between four and five years, and one five to 10 years. Of the moderately advanced group, 28 had treatment for less than two years, 41 between two and four years, 18 between four and five years, and 40 between five and 10 years. Of the far advanced group, 15 took pneumoperitoneum less than two years, 33 between two and four years, 15 from four to five years, and 49 from five to 10 years. Of the 246 reported as having taken the treatment successfully, actually 38 are continuing to take this treatment. Many of these could well have been discontinued for the past three to four years, but due to a reluctance on the part of the patient, and in some cases the Doctor in attendance, it has been continued. However, we have not insisted on discontinuing it. In general, moderately advanced cases should be continued three to four years and far advanced cases from five to six years.

TABLE VI

TIME SINCE PNEUMOPERITONEUM DISCONTINUED: SUCCESSFUL CASES

	Minimal	Moderately Advanced	Far Advanced	Total
Continuing	1	19	18	38
½- 2 years	1	29	22	52
2- 4 years	0	35	37	72
4- 5 years	2	15	8	25
5-10 years	1	29	27	57
TOTAL	5	127	112*	244

*2 F. A. No report on time.

In the failure group, one minimal case took the treatment from two to four, and one from five to 10 years. Of the moderately advanced group, 13 took treatment less than two years, 13 discontinued between the second and fourth year, and five between the fourth and fifth year, and 10 between five and 10 years. Of the far advanced group, 72 discontinued the treatment before two years, 55 took it less than four years, 17 from four to five years, and 27 continued for five to 10 years. The majority of patients with far advanced disease took treatment less than four years.

Of the 246 successful cases, one with minimal disease still continues treatment, one has been discontinued less than two years, two from four to five years, and one five to 10 years. Of the moderately advanced group, 19 are continuing pneumoperitoneum, but most of them could well have discontinued it. Twenty nine have had pneumoperitoneum discontinued from one-half to two years, 35 for two to four years, 15 from four to five years, and 29 from five to 10 years. Of the far advanced group, 18 continue to take treatment, 22 have been stopped between one-half to two years, 37 from two to four years, eight from four to five years, and 27 from five to 10 years.

Although pneumoperitoneum with temporary phrenic nerve paralysis is probably the newest of the temporary collapse measures, it has proved its value. The results of this treatment are comparable, or even better, than other temporary collapse measures, and it is by far the safest procedure in use today. In our series there has not been one case of air embolus. Tuberculous peritonitis has occurred in less than 1 per cent, and these, except one minimal case, have been in the far advanced group. Since this is about the incidence of peritonitis in far advanced cases without any treatment, the question may be posed as to how many of the 337 cases that were far advanced might have developed tuberculous peritonitis without collapse therapy. There are 246 of our cases who are considered as having been successfully treated, which in our opinion makes pneumoperitoneum with phrenic nerve operation, a number-one procedure. This coupled with a small percentage of complications, noted in this series, makes us prefer pneumoperitoneum as a temporary collapse measure.

Pneumoperitoneum does not produce satisfactory results in cases with predominantly productive lesions. If these cases are at all suitable thoracoplasty is preferable.

Large enough fills must be given that the height of the diaphragm on the side where the phrenic crush has been done will reach the third interspace. This requires refills at weekly intervals of 700 to 1,000 cc.

In our institution most patients take modified instead of strict bed rest and thus we do not have a comparable group who have taken only bed rest to compare with our group of collapse cases. However, all of our patients with this type of collapse therapy have had a reasonable amount of bed rest.

Case 1: E. W. was admitted to the hospital November 28, 1944 with moderately advanced tuberculosis: small area of disease with small cavity in the upper lobe on the right lung. Sputum was positive. She had diabetes. Pneumoperitoneum was started December 11, 1944 and she was discharged August 23, 1947 with arrested disease. Pneumoperitoneum was discontinued in February 1949, and as a precautionary measure right phrenic nerve crush was done. X-ray films since discharge have shown stable lesions. This patient is working in our institution full time and is considered arrested.

Case 2: Mrs. J. C. was admitted December 6, 1943 with a diagnosis of pneumonic tuberculosis, far advanced. Her temperature ranged from 101 to 102 degrees F. and her sputum contained tubercle bacilli. Pneumoperitoneum was started December 31, 1943 and she was discharged January 27, 1948. Pneumoperitoneum was continued until May 10, 1948. She has been working in the Sanatorium full time since discharge and all x-ray films have shown stable lesions. Her present status is apparently cured.

Case 3: C. F. B. This 40 year old white male was admitted February 16, 1944 with far advanced pulmonary tuberculosis throughout both lungs with large cavities in the right. Tuberculous laryngitis was also present. His sputum was positive. Pneumoperitoneum was given from March 11, 1944 to August 15, 1949. Right phrenemphraxis was done March 2, 1944. Laryngoscopy in 1947 revealed healed chronic tuberculous laryngitis. The sputum was converted within six months, but since he has had occasional positive culture. Exercise was started in 1946 and although still a patient,

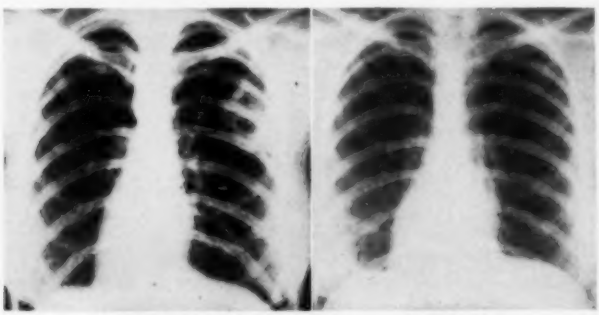

FIGURE 1a FIGURE 1c

Figure 1a: From x-ray film of November 28, 1944, before treatment shows disease with cavity; right second intercostal space.

Figure 1c: From film of January 20, 1950, lesion thoroughly resolved; pneumoperitoneum discontinued as effective.

he is doing seven hours light work per day at the sanatorium. Present x-ray films show evidence of stable lesions. Prognosis is now considered good and present status is arrested.

Case 4: M. N., age 30, was admitted to our institution May 1, 1944. She had far advanced, C, tuberculosis with involvement throughout the right lung and considerable disease in the left. She had multiple cavities in the right, especially at the base. Her sputum was positive. Pneumoperitoneum was instituted May 10, 1944 and continued until July 12, 1948. Her sputum converted after about six months, and the lesions have appeared to be stable for the past one or two years. She is still a patient in the institution, but is doing several hours light work per day. Actually she has been ready for discharge for months, but due to her dread of attempting to lead a normal life, she has been reluctant to accept a discharge. We have classified her tuberculosis as arrested, though actually it may be apparently cured. This is one of the few cases of basal lesions on whom we have obtained good results contrary to the popular conception that such cases are particularly suitable for pneumoperitoneum.

Case 5: C. Y. This patient of 32 years was admitted June 21, 1943 with far advanced pulmonary tuberculosis. She had some involvement of the right lung and a huge cavity in the left mid-lung field. Her sputum was positive. Pneumoperitoneum was started July 17, 1943 and she was discharged February 19, 1944 as improved. At discharge, after only seven months of treatment her sputum was negative and the cavity was apparently closed. Pneumoperitoneum was continued to August 1947, at which time, and since, she has been leading a normal life as housewife. All x-ray films since discharge have shown evidence of stable lesions, or no lesions, and she is at present classified as apparently cured.

Case 6: G. R. This 20 year old white male was admitted May 17, 1942 with pulmonary tuberculosis, far advanced, complicated by tuberculous

FIGURE 6a FIGURE 6c

Figure 6a: X-ray film of May 15, 1942, note dense lesion in upper half of right lung with cavitation and some disease in the left lung with small cavity under left second anterior rib shadow.

Figure 6c: From film of August 6, 1945, evidence of right cavity; stabilized disease on the left; right thoracoplasty was done.

laryngitis. There was disease throughout the upper two-thirds of both lungs with a large cavity in the right apex and a small cavity in the left apex. Right pneumothorax was attempted but was not satisfactory. Pneumoperitoneum was started June 30, 1942 and right phrenemphraxis was done June 25, 1942, resulting in a satisfactory rise of the diaphragm. Sputum remained positive. He was discharged April 2, 1943 to continue treatment at home. He was readmitted November 24, 1944 with far advanced pulmonary disease, but apparently the laryngitis had cleared. Pneumoperitoneum was continued until June 1945, when disease on the left side appeared to be stable and right thoracoplasty was done. In February 1948 the disease in the left lung reactivated and he was readmitted February 24, 1948. Pneumoperitoneum was reinstituted. The disease in the left lung has again stabilized and he is on seven hours exercise. In our opinion, he was successfully prepared for right thoracoplasty. He is still a patient, but will be ready for discharge at an early date. His status at present is arrested.

SUMMARY

We have used pneumoperitoneum in conjunction with phrenemphraxis on the worse side in most of the 560 white men and women, including minimal, moderately advanced and far advanced cases of pulmonary tuberculosis.

Of these 246 have been successfully treated and most of them discontinued pneumoperitoneum in two to 10 years. This procedure is not particularly suitable for minimal cases, nor those with predominantly fibrotic lesions.

This type of temporary collapse has been the orthodox treatment in Georgia for the past hive to six years. In our institution we give an average of about 25,000 fills yearly, with a patient load average of 1,500.

RESUMEN

Hemos usado el enumoperitóneo en combinación con frenopraxis en el lado mas enfermo en la mayoría de 560 hombres y mujeres de raza blanca, incluyendo casos con lesiones mínimas, moderadamente avanzadas y muy avanzadas de tuberculosis pulmonar.

De éstos, 246 han sido tratados con éxito y la mayoría suspendieron el neumoperitóneo entre dos y diez años. Este procedimiento no es especialmente adecuado para los casos mínimos ni para los que tienen lesiones de predominio fibroso.

Este tipo de colapso temporal ha sido el tratamiento ortodoxo en Georgia por los últimos cinco o seis años. En nuestra institución hacemos un promedio de 25,000 reinsuflaciones por año con una media de 1,500 enfermos.

RESUME

Les auteurs ont utilisé le pneumopéritoine associé avec la paralysie phrénique du côté le plus atteint chez plus de 560 hommes

ou femmes de race blanche atteints de tuberculose pulmonaire minime, moyenne ou grave. Parmi ces malades, 246 ont répondu avec succès au traitement et la plupart d'entre eux ont cessé leur pneumopéritoine après deux à dix ans. Cette technique n'est pas à conseiller pour les cas minimes, non plus que pour ceux où les lésions fibreuses sont prédominantes.

Cette méthode de collapsothérapie temporaire a été le traitement considéré comme orthodoxe en Géorgie pendant les derniers cinq ou six ans. Dans leur établissement, les auteurs font en moyenne chaque année 25,000 insufflations pour un total moyen de 1,500 malades.

REFERENCES

1 Banyai, A. L.: "Pneumoperitoneum Treatment," St. Louis, 1946, *The C. V. Mosby Co.*
2 Crow, Horace E. and Whelchel, Fred C.: "Diaphragmatic Paralysis and Pneumoperitoneum," *Amer. Review Tuberc.*, 52:367, 1945.

The Treatment of Intestinal Tuberculosis*

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Since the discovery of antibiotics, numerous authors have observed their effect on intestinal tuberculosis. The conclusions are not uniform. The action of streptomycin has been particularly studied by Sweany,[1,2] Mason,[3] and Hinshaw[4] in the United States, Markoff[5] in Switzerland; we ourselves have communicated our results on the occasion of six observations,[6] and more recently have given general views on this subject.[7]

Para-aminosalicylic acid was the chief subject of works by Swedish authors Carstensen and Sjolin,[8] and Ivar Kallqvist.[9] More recently, T.B./1 claimed the attention of Boehm,[10] and Kulhmann,[11] etc.

Working Scheme

We shall first present 24 observations of intestinal tuberculosis, which were gathered in Bordeaux and Marseilles hospitals, and then show the results of the investigations made among 30 sanatoriums in France.

Observations

Of the 24 patients considered, 23 presented intestinal tuberculosis, secondary to pulmonary tuberculosis. Only one of the patients had isolated intestinal localization of the disease.

The clinical and bacteriological studies, as well as the x-ray films, support the diagnosis.

With regard to radiology, nearly all patients were first examined through an opaque meal, then through a clyster with baryta, and finally, after evacuation, through insufflation of the intestinal tube.

The patients were all treated with streptomycin, intra-muscular, in doses from 1 to 2 grams a day for a period covering one to three months. Three of these patients were given para-aminosalicylic acid treatment.

We have endeavored to classify these cases according to the lesions, as they appeared from the clinical and radiological studies. We thus classified our patients into six groups, corresponding to different lesions and syndromes.

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First Group: Ulcerous Diffuse Tuberculosis of the Intestine.

Nine patients were observed with various pulmonary conditions, serious in most cases. They had profuse diarrhea, abdominal pains and poor general condition. Their emaciation resulted from deficiency in assimilation, or dehydration. Their temperature was high and fluctuating. With all of them a remarkable effect of streptomycin on the intestinal lesions and on the general condition was observed.

Two of these patients had complete cleaning of their pulmonary lesions. Two others were able to undergo surgical collapse. In three others the improvement of pulmonary lesions was moderate, without collapse, and the remaining two died, one of cachexia with phlebitis, and the other from hemoptysis. Autopsy of the latter, six months after the clinical healing of his intestinal troubles, disclosed the existence of numerous cicatrized ulcerations in the ileum and the large intestine.

One patient was treated solely with para-aminosalicylic acid for two months, in doses of 14 grams a day when the diarrhea and emaciation were worse, with total loss of appetite. With two months of streptomycin the digestive troubles ended, making the x-ray pictures more normal, and the weight increased 8 kilograms.

After periods of from six months to two years, none of the nine cases had relapsed.

Second Group: Localized Ileo-caecal Tuberculosis.

These five patients were treated with streptomycin longer than those of the first group. One of them subsequently was given para-aminosalicylic acid. These presented various troubles, chiefly diarrhea, with interruptions at times of pseudo occlusive phenomena. In the right iliac fossa, bulky caecal tumors could be felt.

Four patients healed within one to three months of streptomycin administration from clinical and radiological evidence. Only one patient, after receiving antibiotic for one month, had to be operated upon, notwithstanding clinical improvement. The removed specimen showed scar with fibrous and retractile reaction which permits considering this case as complete healing.

Third Group: Tuberculosis with Stenosis of the Small Intestine.

These three cases presented various phenomena such as diarrhea, alternating with constipation. Evidence of obstruction was the predominant symptom. These patients were in poor general condition. Streptomycin was administered in usual doses, and one received para-aminosalicylic acid, which was discontinued for lack of tolerance.

In spite of the drugs, the strictures gradually grew tighter and dehydration of the patients grew worse. Two had to undergo surgery.

The removed stenosed areas (one in one case and eight in the other) revealed the exact nature of inflammatory shrinkings, brought about by recent tuberculous lesions. These patients experienced return of appetite, increase in weight, etc. At the same time, pulmonary improvement was so considerable that healing seemed possible.

The third patient, because of phlebitis and a most serious general condition, could not be operated upon. Autopsy disclosed the existence of three stenoses with ulcerations. On the other hand, numerous ulcerations on the remainder of the small intestine and of the colon could be seen microscopically and histologically as being healed.

Fourth Group: Intestinal Tuberculosis with General Granulation.

The three cases with this disease died, notwithstanding the use of antibiotics.

Fifth Group: Isolated Rectal Tuberculosis.

This group consisted of three cases, of which two were observed by Cattan.[12] All of them were successes for streptomycin within a period of 30 to 45 days.

Primitive Intestinal Tuberculosis

Finally, one case of "primitive" intestinal tuberculosis was observed in a patient presenting a cutaneous allergy to tuberculin, tuberculous epididymis, and cutaneous tuberculosis. The stools contained tubercle bacillus, and radiological examination confirmed the suspicion of ileo-caecal tuberculosis. One gram of streptomycin was administered daily for five months and no symptom has occurred for one and a half years.

Investigations at Sanatoriums

We have made investigations in 30 sanatoriums with the conclusion that *ever since the use of streptomycin, intestinal tuberculosis is extremely rare in France.* Those establishments which previously always had several cases of intestinal tuberculosis have had from none to a very small number during the last three years.

Several doctors cling to the old methods: vitamin therapy, physiotherapy, surgery. Most phthisiologists who have observed cases of intestinal tuberculosis treated with streptomycin agree that the results are "spectacular."

Discussion

In this debate on the value of the various antibiotics towards intestinal tuberculosis, we are anxious to present these 24 cases to the credit of streptomycin.

Thiosemicarbazones do not appear to be without danger, as sanguine and hepatic troubles are reported by numerous authors. The effect seems to be slower and inferior to that of streptomycin.

Para-aminosalicylic acid was not tolerated in two cases, and it was necessary to stop the treatment. In one case, after two months, the condition of the patient became worse, and streptomycin had to be used. Judging from the studies of Swedish authors, the frequency of digestive intolerances to para-aminosalicylic acid is considerable with persons having intestinal tuberculosis. Moreover it requires several months before results appear.

During the course of intestinal tuberculosis which rapidly makes for an extremely weak general condition, streptomycin seems to work more quickly, sometimes stopping diarrhea in three to four days and increasing weight by several kilograms within two to three weeks. This fact alone would be sufficient to give preference to this drug. No toxic manifestation occurred in any of our 24 patients.

In the presence of an ulcerous diffuse form of tuberculosis localized in the ileum and caecum, much can be expected from the antibiotic. On the contrary, in the case of stenosis in the small intestine, surgical intervention must be contemplated.

At present, thanks to antibiotics, intestinal tuberculosis in a phthisical person no longer means certain death, and does not contraindicate collapse therapy.

SUMMARY

1) Twenty-four cases of intestinal tuberculosis have been treated by streptomycin with generally good results, but variable according to the anatomic form of lesions.

2) The ulcerous diffuse forms, the isolated ileo-caecal and rectal lesions, respond to the antibiotic.

3) In the stenosis form in the small intestine, streptomycin constitutes a good preoperatory treatment and auxiliary, but does not prevent radical intervention.

4) "Primitive" intestinal tuberculosis seems to be influenced favorably by the antibiotic.

5) Since the use of antibiotics, the frequency of intestinal tuberculosis has diminished considerably in France.

6) As compared to para-aminosalicylic acid and to amithiozone,

the action of which seems to be slower and not so well tolerated, streptomycin appears to deserve first place.

7) Thanks to streptomycin, the influence of intestinal tuberculosis on pulmonary lesions of phthisical persons loses its dreadful meaning of aggravation and enlarges the limits of collapse therapy.

RESUMEN

1) Se han tratado veinticuatro casos de tuberculosis intestinal por la estreptomicina con resultados generalmente favorables pero variables de acuerdo con la forma anatómica de las lesiones.

2) Las formas ulcerosas difusas, las aisladas ileocecales y las rectales responden al antibiótico.

3) En la forma estenosante del intestino delgado la estreptomicina es un buen tratamiento properatorio auxiliar pero no evita la intervención radical.

4) La tuberculosis intestinal "primitiva" parece ser influenciada favorablemente por el antibiótico.

5) Desde que se emplean los antibióticos la frecuencia de la tuberculosis intestinal ha disminuído considerablemente en Francia.

6) En comparación con el ácido paraminosalicílico y con la amithiozona, de la cual la acción parece ser mas lenta y no tan bien tolerada, la estreptomicina parece merecer el primer lugar.

7) Gracias a la estreptomicina la influencia de la tuberculosis intestinal en las lesiones pulmonares tuberculosas pierde su temible significación de gravedad y amplía las posibilidades de la colapsoterapia.

RESUME

1) 24 cas de tuberculose intestinale ont été traités par la streptomycine avec, en général, de bons résultats, variables cependant suivant la forme anatomique des lésions.

2) Les formes ulcéreuses diffuses, les lésions iléo coecales et rectales isolées répondent à l'antibiotique.

3) Dans les formes sténosantes de l'intestin grêle la streptomycine constitue un bon traitement pré-opératoire et adjuvant, mais ne dispense pas de l'intervention radicale.

4) La tuberculose intestinale primitive semble être influencée favorablement par l'antibiotique.

5) Depuis l'emploi des antibiotiques la fréquence de latuberculose intestinale a considérablement diminué en France.

6) Comparée au PAS et à l'amithiozone, dont l'action semble plus lente et moins bien tolérée, la streptomycine semble mériter la première place.

7) Grâce à la streptomycine, l'influence de la tuberculose intestinale sur les lésions pulmonaires des phtisiques perd sa terrible signification d'aggravation et élargit les limites de la collapsothérapie.

REFERENCES

1 Sweany, H. C.: "Streptomycin in Tuberculous Enteritis," *Am. Rev. Tuberc.*, 56:415, 1942.
2 Sweany, H. C., Lichtenstein, M. R., Turber, C. G. and LeRoy: "Streptomycin Treatment of Tuberculous Enteroolitis: Results in 30 Cases," *Am. Rev. Tuberc.*, 60:576, 1949.
3 Mason, E., Kridelbauge, W. W., Crouch, W. H. and Ward, M.: "Streptomycin in the Treatment of Tuberculous Enteritis: A Report of 33 Cases," *Am. J. Med. Science*, 217:28, 1949.
4 Hinshaw, H. C. and Feldman, W. H.: "Streptomycin, a Valuable Antituberculosis Agent," *Brit. Med. J.*, Jan. 17:87, 1948.
5 Markoff, N.: "Die Streptomycinbehand der Darmtuberkulose," *Sweiz Med. Wschr.*, 78:329, 1948.
6 Damade, R., Piechaud, F., Traissac, F. J., Freour, P. and Roux, M.: "Efficiency of Streptomycin in Intestinal Tuberculosis," *Sté. Francaise de la Tuberculose*, sitting of Jan. 8, 1949. *In Rev. de la Tuberc.*, 13: 335, 1949.
7 Roux, M.: "Intestinal Tuberculosis: Anatomic, Clinical and Therapeutic Study," Thesis, Bordeaux, 1950.
8 Carstensen, B. and Sjolin, S.: "Para-aminosalicylic Acid in Pulmonary Tuberculosis with Secondary Intestinal Tuberculosis," *Svenska Lakartidningen*, 45:729, 1948.
9 Kallqvist, I.: "Para-aminosalicylic Acid in Intestinal Tuberculosis," *Am. Rev. Tuberc.*, 61:621, 1950.
10 Boehm, P.: "A Stride in the Mechanism of Healing of Tuberculous Processes of the Intestine Unser Chamtherayp," *Acta Tuberc. Scand.*, 30, 1950.
11 Kulmann, F.: "Die Behandlung der Darmtuberkulose mit T 61/698 E," *Deutsche Gesund-keitswesen*, 5-6:217, 1949.
12 Cattan, B., Frumusan, P. and Cousin, R.: "Digestive Tuberculosis and Streptomycin," *Arch. de mal. de l'appareil dig.*, 38:1180, 1949.

Candida (Monilia) Infection of the Respiratory Tract*

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Following Castellani's[1] description of the "tea tasters cough" an extensive literature appeared on the significance of the yeasts in infections of the respiratory tract (Dodge,[2] Orie[3]). This has not, so far, solved the problems.

Though Pijper,[4] Shrewsbury[5] and Robertson[6] deny the existence of bronchomoniliasis, there exists a great deal of recent information, describing such cases. The authors of these case studies entertain little or no doubt about the etiologic connection between the diseases of the respiratory tract and the associated yeasts (Keiper,[7] Valada,[8] Tornell, [9] Kostman,[10] Brown,[11] Greer,[12] Skinner,[13] Conant[14] c.s.). A few points, arising from this problem, might be further examined in order to check the arguments which are brought forward in favor of them.

Microorganisms Involved: Yeasts may either be classified amongst the other fungi (Castellani,[1] Vuillemin,[15] Dodge[2]) or separately. For clinical purposes I prefer the latter (Martin et Jones[16] c.s., Langeron et Guerra[17]).

They are microorganisms, which as a rule appear as round or oval cells and propagate by budding and sometimes by septation. The colonies on artificial culture media, on which at 37 degrees C. as well as at 18 degrees C. growth usually takes place in a few days, mostly have the aspect of smooth, white to light yellow, somewhat moist colonies with usually a pleasant characteristic smell (yeast odor). But there are also dull, in some species sometimes wrinkled colonies, surrounded by much mycelium.

Most yeasts of clinical importance (Candida albicans, Torulopsis neoformans) belong to the Fungi imperfecti and to the subdivision Hyphomycetes and a few to the Ascomycetes, such as Debaryomyces. In addition other fungi may assume the yeast-like form under certain conditions, among these are Blastomyces, Histoplasma (Tables I, II, III, IV).

The indentification of yeasts is based on the presence or absence of (pseudo-) mycelium, and the ability of fermenting carbohydrates, while the pathogenic ones, as far as known, mostly are

*Presented at the First International Congress on Diseases of the Chest of the American College of Chest Physicians, Rome, Italy, September 17-22, 1950.

devoid of sexual spores (ascospores) which the industrial species often do possess. Besides, other specific characteristics are found. Candida albicans for example does form mycelium and ferments glucose and maltose with gas-production, while Torulopsis neoformans is devoid of both characteristics, and is distinguished by its shiny mucoid appearance. Moreover the cells are round or short-oval and nitrate is not assimilated. It should, however, be considered, that besides these two, several related and similar species occur (Stelling Dekker,[18] Lodder,[19] Diddens et Lodder,[20] Langeron et Guerra,[17] Stovall et Buboiz,[21] Martin et Jones,[16] Benham et Hopkins[22]). The yeasts as a rule grow easily on acid (or also neutral), natural and artificial, carbohydrate-containing culture-media (acid malt agar or the Sabouraud agar). The acid malt agar has the advantage of preventing the growth of bacteria to a large extent.

ORDO	FAMILY	SUBFAMILY a.o.	SUBDIVISION a.o.	GENUS a.o.
HYPHOMYCETES	MUCEDINACEAE	I. HYALOSPORAE	GOSPORIEAE	MONILIA
				GEOTRICHUM
			ASPERGILLEAE	ASPERGILLUS
				PENICILLIUM
			BOTRYTIEAE	ACREMONIUM
		II. HYALODIDYMAE		SPOROTRICHUM
	DEMATIACEAE	I. PHAEOSPORAE	PERICONIEAE	ACROTHECA
			TRICHOSPORIEAE	TRICHOSPORIUM
				RHINOCLADIUM
			HAPLOGRAPHIEAE	HORMODENDRON
		III. PHRAGMOSPORAE	ACROTHECIEAE	ACROTHECIUM
		IV. PHAEODICTYAE	ALTERNARIEAE	ALTERNARIA

TABLE I

Phycomycetes				
	Oomycetes			
	Zygomycetes			
Mycomycetes				
	Ascomycetes a.o.	Hemiascineae a.o.		Protomyces
		Exoascineae a.o.		Saccharomycetaceae
				Endomycetaceae
Basidiomycetes				
Fungi Imperfecti	Sphaeropsideae			
	Melanconieae			
	Hyphomycetes	Mucedineae a.o.		Mycotoruloideae
		Dematiaceae		Torulopsidoideae
		Stilbaceae		
		Tuberculariaceae		

TABLE II

For the technics of cultures and media (Stelling Dekker,[18] Diddens et Lodder,[20] Lodder[19]), fermenting (laws of Kluyver), the formation of mycelium (Rivalier et Seydel,[23] Martin[16] c.s.) the formations of spores (Stelling Dekker,[18] Langeron[17] l.c., Stovall et Bubolz[21]) as well as the further methods of differential diagnosis, reference is made to the respective technical articles.

Occurrence of Candida Albicans Outside of Human Body.

Candida albicans does not occur outside the human body, and according to Benham,[24] Guerra,[25] is not even found on the skin of normal persons (Table V). Therefore, the ideas of Ikeda[26] and later the observations of Tornell[9] ascribing albicans infections to dust need further confirmation. Our observations on the air in the Netherlands in the spring and summer of 1947 failed to show the presence of pathogenic yeasts. Of approximately 5,000 fungus colonies found, only 90 were yeasts and none of them belonged to the Candida albicans type, or were of the Torulopsis neoformans species[27] (Table VI).

YEASTS		FAMILY	SUB-FAMILY	
A. SEXUAL SPORULATION (ASCOSPORES)		ENDOMYCETACEAE		
	MYCELIUM ONLY		EREMASCOIDEAE	
	MYCELIUM AND OIDIA		ENDOMYCOIDEAE	
	MYCELIUM WITH CONIDIA (OCCASIONALLY OIDIA) OR BUDDING YEAST CELLS.		SACCHAROMYCOIDEAE	SACCHAROMYCES SP
	MYCELIUM WITH BUDDING YEAST CELLS. SPINDLE-SHAPED SPORES.		NEMATOSPOROIDEAE	DEBARYOMYCES SP.
B. NO SEXUAL SPORULATION				
	I. PRODUCTION OF CAROTINOID PIGMENT.	RHODOTORULACEAE		
	II. NO CAROTINOID PIGMENT.	TORULOPSIDACEAE		
	PSEUDOMYCELIUM ABSENT OR RUDIMENTAL NO "APPAREIL SPORIFERE"		TORULOPSIDOIDEAE	
	(PSEUDO) MYCELIUM WITH BUDS AT THE NODES.		MYCOTORULOIDEAE.	

TABLE III

TORULOPSIDOIDEAE	TORULOPSIS BERLESE	FERMENTING a. o.	TORULOPSIS HOLMII
		NON FERMENTING a. o.	TORULOPSIS NEOFORMANS (SANFELICE) LODDER
	PITTROSPORON SABOURAUD.		
	MYCODERMA FERMENTANS EMEND LEGENDRE.		
	KLOECKERA JANKE.		
MYCOTORULOIDEAE	CANDIDA BERKHOUT EMEND LANGERON GUERRA.		CANDIDA ALBICANS
			CANDIDA TROPICALIS
			CANDIDA PARATROPICALIS
			CANDIDA GUILLIERMONDII
			CANDIDA KRUSEI
			CANDIDA BRUMPTII
			CANDIDA ZEYLANOIDES
	BRETTANOMYCES KUFFERATH ET v. LAER EMEND CUSTERS. ONLY SAPROPHYTIC (BEER AND ALE)		
	TRICHOSPORON BEHREND EMEND DIDDENS ET LODDER a. o.		TRICHOSPORON CUTANEUM

TABLE IV

Pathogenicity: Candida albicans is as a rule stated to be the cause of the lung diseases by yeasts. Many of the exceptions published are of doubtful nature. The pathogenicity of Candida albicans in contrast with other similar species has been conclusively demonstrated by de Minjer,[28] and Mackinnon.[29] To establish the relationship of yeasts to pulmonary infections in man two things are required:

1) that yeasts are adequately identified, and
2) that the methods of isolation permit the evaluation of the result.

It may be taken for granted that yeasts, just like other pathogenic microorganisms are frequently found in the mouth and throat. The investigations of Epstein,[30] Stovall et Bubolz,[21] Todd,[31] Benham et Hopkins[22] and our own fully confirm this point. Together with other authors (a.o. Greer[12]) it is our opinion that most of the "monilia-infections" result from a false interpretation of sputum slides and cultures. The yeasts do not originate from the lungs, but are added to the sputum in the oral cavity. Contrary to others we are of the opinion that if one wants to eliminate the influence of this oral flora on the results of the examination of the sputum, only two methods are at our disposal:

1) the direct needle aspiration of the lung (Bullowa[32]), and
2) the application of the method worked out by Mulder,[33] consisting of repeated washing of the sputum in saline until the adhesive mucus is removed.

If one does use one of the two methods, yeast cells are rarely found. If one does not, they are commonly seen in the sputum. The bacteriology of bronchoscopically removed sputum seems to be unreliable considering our own observations. During prelim-

TABLE V

SOURCE OF SOME IMPORTANT YEASTS (Diddens et Lodder)

	PARASITIC i.e. human or animal origin.	SAPROPHYTIC i.e. from dead material.
Candida albicans	79	0
Candida Krusei	17	17
Candida tropicalis	23	12
Candida zeylanoides	5	1
Candida melinii	0	9
Brettanomyces sp.	0	9
Trichosporon pullulans	0	13
Trichosporon cutaneum	11	7

inary observations we found no yeasts in blood cultures, pleural fluid and urine. (In the latter a few exceptions). From the mouth of 31 patients in 17 cases yeasts were grown.

Candida albicans	6
Candida zeylanoides	4
Candida Krusei	1
Candida species	1
Debaryomyces Matruchoti	3
Other species	2

From 10 healthy people Candida albicans was isolated three times and Torulopsis sp. were not found. Thus in 50 per cent of the control cases yeasts were grown. In healthy people the number of colonies as well as the percentage of cases seems to be a little lower. Rinsing of the mouth does not alter the figures substantially.

Subsequently we investigated a number of cases in which lung disease of some kind was present (about 50 per cent tuberculous), not yet aware of the importance of proper handling of the sputum.

In a large number of cases yeast cells were seen in the sputum slides (Gram stain) as well as in the cultures (Table VII and the first part of Table VIII). After that we realized that maybe these incomprehensible results were due to the method of examination, and the figures given in the second part of Table VIII are the results of approximately 2,000 sputa after the method of Mulder had been introduced.

TABLE VI

FUNGI FROM THE AIR (February-September, 1947) GRONINGEN

Actinomyces	sp.	80
Yeasts		90
Geotrichum	sp.	5
Aspergillus	sp.	360
Penicillium	sp.	± 500
Sporotrichum	sp.	15
Sepedonium	sp. (?)	5
Botrytis	sp.	420
Hormodendron	sp.	±5000
Alternaria	sp.	35
Stemphylium	sp.	185
Fusarium	sp.	45
Different		285
TOTAL		7025

1 per cent Yeasts, no Candida (Monilia) Albicans.

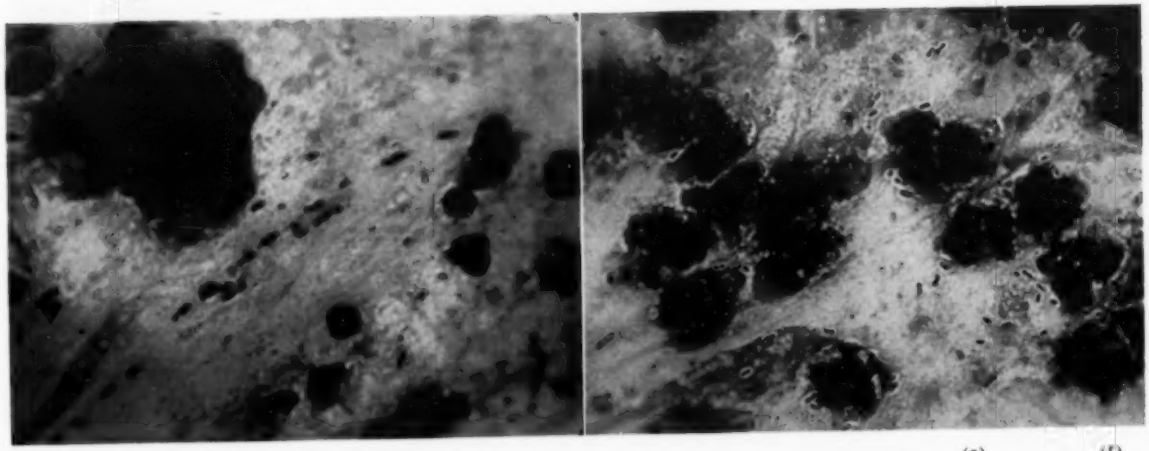


FIGURE 1

(a) Polygonal-buccal-epithelial cell.
(b) Polynuclear leucocytes.
(c) Yeasts.

FIGURE 2

(d) Pneumococci.
(e) Pneumococci.
(f) Polynuclear leucocytes.

It will be clear from the figures in Table VIII that we decided to group this first series of sputum results together with the mouth examinations under the heading of "control group from the oral cavity."

The following facts substantiate the reliability of these theories and observations and give support to the concept that the lower air tract is normally sterile and demonstrate at the same time that infection apparently is as a rule monobacterial:

1) The results of the data found by aspiration of a small amount of lung fluid as introduced by Bullowa,[32] and the

2) nearly always sterile findings in the Monaldi drainage fluids on first examination (except, of course, tubercle bacilli).

3) Results of puncture of lungs removed operatively.[34]

4) The fact, that findings obtained this way may be duplicated on successive days.

5) The fact, that the disappearance of oral polygonal epithelial cells goes together with the disappearance of the "oral" flora, if one makes consecutive slides during the procedure of washing. The presence of epithelial cells of this type almost always means that bucco-pharyngeal mucus is still present.

6) The close relationship existing between clear-cut clinical en-

TABLE VII

YEASTS FOUND IN THE ORAL CAVITY

	Cultures Cases	Positive	± 1-10 Colonies	Negative	Slides Cases	Positive	1-10 Cells	Negative
Tuberculous sputa	81	38	14	31	53	5	3	45
Non tuberculous sputa	66	23	15	28	59	10	5	44
Total	147	61	27	59	112	15	8	89
Controls	41	—	20	21				
"patients" + controls = control group from the oral cavity	188	61	47	80	112	15	8	89

TABLE VIII

YEASTS FREQUENTLY FOUND IN THE ORAL CAVITY COMPARED TO OCCURRENCE IN PROPERLY WASHED SPUTUM

Oral cavity "patients" + controls *Before*	Cultures cases 188	+ 61	± 47	— 80	Slides cases 112	+ 15	± 8	— 89
Sputum *After* washing of sputum	30	3	0	27	±2000	3	17	±1980

tities (bronchiectasis, lobar pneumonia, tuberculosis [own observation unpublished], eosinophilic bronchial catarrh) and corresponding flora (Haemophilus influenzae [Mulder[33]], lower type pneumococci, and no flora in the two latter conditions). In tuberculosis we found only in 15 per cent of the cases a "secondary" flora—mostly Haemophilus influensae—probably due to coexisting bronchiectasis. Bronchiectasis in tuberculosis, however, gives rise to a Haemophilus bronchitis in only a minority of the cases.

7) The efficiency of therapy corresponding to the sensitivity of the aetiologic flora (Mulder,[33b] Lopez Cardozo[35] c.s.).

8) The complete divergence of the importance ascribed by different authors to different organisms. Yeasts (afore-mentioned authors), Spirochaetes (Smith[36]) and Actinomyces (Kay[37]) are found and considered important by some authors in a large percentage of cases and are considered to be devoid of importance by many others of equal authority. (The photographs shown in Figures 1 and 2 will make clear the difference between sputum before and after treatment).

We do not at all claim this to be a new method. Several authors have described related techniques (Smith[36]). Prof. Mulder, Leiden University, has worked it out as a practicable method, and it has been confirmed by others (Lubsen[38]). We only show its application in the controversial field of moniliasis.

Discussion

The pathogenetic power of certain yeasts is well established for animals. Generalized cases prove the same for man. Pathogenic yeasts do not occur in the air. "Dust infections" are consequently extremely improbable.

Occurrence of Candida albicans in the mouth is frequent, the presence of yeasts in sputum on the other hand is rare. If not, there is usually a faulty technic. It must be evident that the sputum originated from the deeper air passages. Unless pulmonary aspiration biopsy is performed, adequate washing of the sputum is required.

The presence of polygonal epithelial cells in the sputum slide makes an admixture of buccal mucus extremely probable. As a rule only adequate washing (Mulder) or aspiration biopsy gives us the required accurate sputum results. This basic fact is substantiated by many arguments.

We ourselves, during eight years' experience have seen only three transient cases of bronchopulmonary yeast infection and are unable to give a clinical description of the disease. We now believe that a clinical entity can not be given and in the rare cases a diagnosis must be made by biopsy, sputum examination or autopsy.

Yeasts sometimes invade the respiratory tract in cases of breakdown of the general resistance or of failure of the expectoration (narcosis). Most cases probably occur on the basis of an underlying debilitating disease.

In those cases in which the invasion in the lower air passages is an established fact, a serious course of the disease is repeatedly described. Also, less serious forms, and even mild ones are known, as we ourselves observed.

Lung infections by yeasts are probably partly also a result of hematogenic spread. In those cases a negative sputum examination of course, does not exclude an infection.

If the above mentioned criteria are strictly observed, we think that "bronchomoniliasis" will be again considered as a rare disease and often as a secondary type of infection as Pijper supposed it was, 25 years ago.

SUMMARY

1) Pathogenic yeasts occur. One of the most important, Candida (Monilia) albicans, however, does not occur outside the human body.

2) "Dust infections" with Candida albicans are consequently dubious.

3) Candida albicans is ubiquitous in the mouth.

4) Candida albicans is rarely found in the sputum, contrary experiences are due to faulty technic or misinterpretation of sputum slides.

5) Bronchopulmonary moniliasis (Candida albicans infection) is a rare disease.

Acknowledgment: The author is very much indebted to Prof. Joha Westerdgk and Dr. A. V. Beverrvyk, Baarn, Holland, for much assistance and helpful criticism in the mycological problems.

RESUMEN

1) Existen levadura patogénas. Una de las mas importantes la Candida (Monilia) albicans, sin embargo no se encuentra fuera del cuerpo humano.

2) Las "Infecciones por el polvo" con la cándida albicans son por consecuencia dudosas.

3) La cándida albicans es huesped de la boca.

4) Raras veces se encuentra en el esputo, las experiencias contrarias a esto se deben a falta de técnica o a mala interpretación de los frotis de esputos.

5) La moniliasis broncopulmonar es una enfermedad rara.

RESUME

1) Parmi les champignons pathologiques, un des plus importants, le Candida albicans, ne se rencontre pas en dehors du corps humain.

2) Par conséquent, le rôle des poussières dans l'infection à monilia doit être mis en doute.

3) Candida albicans est un hôte habituel de la bouche.

4) Candida albicans se trouve rarement dans les crachats. Les opinions contraires relèvent de fautes de techniques ou de mauvaise interprétation des lames où ont été étalés les crachats.

5) La moniliase broncho-pulmonaire (infection à Candida albicans) est une affection rare.

REFERENCES

1 Castellani, A.: "Tropical Blastomycosis," *Philippine Jour. Sc.*, ¿?:197, 1910.
2 Dodge, C. W.: "Fungous Diseases of Man and Other Mammals," Mycology, *Mosby*, St. Louis, 1935.
3 Orie, N. G. M.: Thesis Utrecht, 1946.
4 Pijper, A.: "Bronchomoniliasis and Monilia Fungi Found in Sputum," *M. J. South Africa*, 19:101, 1925.
5 Shrewsbury, J.: "Secondary Thrush of the Bronchi," *Quart. J. Med.*, 5:375, 1936.
6 Robertson, R. F.: "Pulmonary Moniliasis," *Edinburgh M. J.*, 55:274, 1948.
7 Keiper: "Studies on Yeast-like Fungi Isolated from Pulmonary Disease (Bronchomoniliasis)," *J. Lab. and Clin. Med.*, 23:343, 1938.
8 Valada, H. J.: "Broncho-pulmonary Mycosis," *Ann. Otol., Rhin. and Laryng.*, 49:289, 1940.
9 Tornell, E.: "Thresher's Lung," *Acta Med. Scand.*, 125:191, 1946.
10 Kostmann, R.: "Moniliasis of the Respiratory Tract," *Acta Paediatrica*, 32:520, 1945.
11 Brown, T. G.: "Pulmonary Mycosis," *Edinburgh M. J.*, 53:583, 1946.
12 Greer, A. E.: "The Synergism Between Mycotic and Tuberculous Infections of the Lungs," *Dis. Chest*, 14:33, 1948.
13 Skinner, C. E.: "The Yeast-like Fungi: Candida and Brettanomyces," *Bact. Review*, 11:227, 1947.
14 Conant, N. F.: "Manual of Clinical Mycology," *Saunders*, Philadelphia, 1944.
15 Vuillemin, P.: "Les champions parasites et les mycoses de l'homme," Paris, *Chevalier*, 1931.
16 Martin, S., Jones, P. C., Yao, K. F. and Lee, L. E.: "A Practical Classification of the Monilias," *Jour. of Bact.*, 34:1, 1937.
17 Langeron, M. and Guerra, R.: "Nouvelles recherches de Zymologie medicale," *Annales de Parasitologie*, No. 1, 2, 5, 3, 1938.
18 Stelling Dekker, N. M.: "Die sporogenen Hefen," *Thesis Utrecht*, 1931.
19 Lodder, J.: "Die anascosporogenen Hefen," Erste Halfte, *Thesis Utrecht*, 1934.
20 Diddens, H. A. and Lodder, J.: "Die anaskosporogenen Hefen," Zweite Halfte, *N. V. Noord-Hollandsche Uitgevers My*, Amsterdam, 1942.
21 Stovall, W. D. and Bubolz, A.: "Identification of Certain Pathogenic Fungi for Men," *Am. J. Public Health*, 22:493, 1932.
22 Benham, R. W. and Hopkins, A. M.: "Yeast-like Fungi Found on the Skin and in the Intestines of Normal Subjects," *Arch. Derm. and Syph.*, 28:532, 1933.
23 Rivalier, E. and Seydel, S.: "Cultures minces sur lames gelosées, colorées et examinées in situ en préparations definitives pour l'étude des cryptogames microscopiques," *Comptes Rendues Soc. Biol.*, 150:181, 1932.

24 Benham, R. W.: "Certain Monilias Parasitic on Man," *J. Inf. Dis.*, 49: 183, 1931.
25 Guerra, P.: "Role des levures en dermatologie," *Thesis Fac. Med.*, Paris, 1935.
26 Ikeda, K.: "Monilia Infections of the Lungs (Bronchomoniliasis)," *Am. J. Clin. Path.*, 7:76, 1937.
27 Orie, N. G. M.: To be published in Antonie van Leeuwenhoek, Amsterdam.
28 Minjer, A.: *Thesis Utrecht*, 1941.
29 Mackinnon, J. E.: "Caracteres y grado de virulencia experimental las Torulopsidaceas de la sub-familia Mycotoruleas (Monilias)," *Ann. de la Fac. de Med.*, Montevideo, 21:5, 6, 7, 1936.
30 Epstein, B.: "Studien Zur Soorkrankheit," *Jahrbuch fur Kinderheilkunde*, 104:129, 1924.
31 Todd, R. L.: "Studies on Yeast-like Organisms Isolated from Mouth and Throat of Normal Persons," *Am. J. Hyg.*, 25:212, 1937. Ref., *J.A.M.A.*, 108:1831, 1937.
32 Bullowa, J. G. M.: "The Management of the Pneumonias," London, 1937.
33 Mulder, J.: *Thesis Groningen*, 1937.
33a Mulder, J.: "Haemophilus Influensae (Pfeiffer) as an Ubiquitous Cause of Common Acute and Chronical Purulent Bronchitis," *Acta. Med. Scand.*, 94:98, 1938.
33b Mulder, J., v.d. Plas, M. C. and Lopes Cardozo, P.: "On the Treatment of Bronchial Infections," *Ned. Alg. Ziektekundige Vereniging*, Session of April 1, 1950.
34 Unpublished observations.
35 Lopez Cardozo, P., v.d. Plas, M. C. and v.d. Ban, M.: "Uitkomsten der behandeling van etterige bronchitis," *Ned. Tijdschrift v. Geneeskunde*, 94:739, 1950.
36 Smith, D.: "Oral Spirochaetes and Related Organisms in Fuso-spirochaetal Disease," *Baillere, Tindall and Cox*, London, 1933.
37 Kay, E. B.: "Bronchopulmonary Actinomycosis," *Ann. Int. Med.*, 26: 581, 1947.
38 Lubsen, N.: "De betekenis van het bacteriologisch sputumonderzoek voor de diagnostiek en de therapie van longziekten," *Scheltema en Holkema*, Amsterdam, 1948.

ERRATUM NOTICE

In the article by Dr. John H. Aldes, entitled "Treatment of Abscesses Associated with Spinal Tuberculosis," published in the May 1952 issue, in figure 6 the pathology was not clearly shown in the illustrations. In the original illustrations the pathology by means of diotrast studies in the lateral and A.P. x-ray views were to show the extension of sinus tract from region of L2 and L3 to the lesser trochanter of femur.

College Chapter News

ARIZONA CHAPTER

The Arizona Chapter of the American College of Chest Physicians announces the election of the following officers during the meeting held in May: President, Joseph N. Plumer, Tucson; Vice-President, Albert Eckstein, Phoenix; Secretary-Treasurer, Leslie B. Smith, Phoenix. Guest speakers were Doctors Edwin R. Levine and William M. Lees of Chicago.

FLORIDA CHAPTER

The Florida Chapter met at Hollywood, Florida, in conjunction with the Annual Meeting of the Florida State Medical Society. The following officers were elected: President, Alexander Libow, Miami Beach; Vice President, Nathaniel Levin, Miami; Secretary-Treasurer, DeWitt Daughtry, Miami; Chairman of the Program Committee, Hawley Seiler, Tampa.

MINNESOTA CHAPTER

The annual meeting of the Minnesota Chapter of the American College of Chest Physicians was held at the Radisson Hotel, Minneapolis, May 26. An election of officers was held, and Dr. Herman Moersch was elected to serve as president for 1952-1953; Dr. A. M. Olsen was re-elected as secretary-treasurer.

Following the business meeting, a scientific program was presented at the Minneapolis Auditorium with Dr. John F. Briggs, presiding.

"Segmental Resections in the Treatment of Pulmonary Infections,"
W. Robert Schmidt.

"Roentgen Observations on Vascular Lesions of the Lung,"
Leo Rigler.

"Treatment of Common Chest Emergencies,"
Thomas J. Kinsella.

"Interrelationships Between Cardiac and Pulmonary Diseases,"
Richard F. Ebert.

A. M. Olsen, Secretary-Treasurer.

MISSOURI CHAPTER

The Missouri Chapter of the American College of Chest Physicians held its annual meeting on March 30, 1952 at the Jefferson Hotel, St. Louis. The Scientific program was presented jointly with the American Trudeau Society.

Round table discussion, "Valvulotomy,"
D. B. Flaven, Chairman.
C. Rollins Hanlon, Thomas Burford, B. C. Bercu, panel members.

"Cineradiography in the Study of Thoracic Lesions,"
Osler Abbott, Atlanta, Georgia.

"Antracosilicosis,"
Louis L. Friedman, Birmingham, Alabama.
Discussion by Emmett F. Pearson, Springfield, Illinois.

"Azotemic Edema of the Lung and Its Relationship to Collagen Disease,"
L. R. Sante, St. Louis, Missouri.
Discussion by Stanley Hampton, St. Louis, Missouri.

NEW ENGLAND CHAPTER

The New England Chapter of the American College of Chest Physicians met at the Deaconess Hospital, Boston, on May 21, 1952. The following officers were elected for the coming year: President, Herbert A. Boyle, Bedford, Massachusetts; Vice-President, Frank Merlino, Providence, Rhode Island; Secretary-Treasurer, John B. Andosca, Boston, Massachusetts.

The speaker of the day was Dr. Philip Schwartz, who spoke on "The Role of Intrathoracic Lymph Nodes in the Pathogenesis of Pulmonary Tuberculosis."

NEW JERSEY CHAPTER

The New Jersey Chapter of the American College of Chest Physicians held its Annual Meeting at Atlantic City, May 20, 1952, in conjunction with the Annual Meeting of the New Jersey State Medical Society. Dr. Martin Sokoloff, Associate Professor of Medicine, Jefferson Medical College, Philadelphia, spoke on the "Recent Advances in Our Knowledge of Pulmonary Empysema." The following officers were elected for 1952-1953: President, John E. Runnels, Scotch Plains; President Elect, J. Earle Stuart, Plainfield; Vice President, Irving J. Selikoff, Patterson; Secretary-Treasurer, Samuel Cohen, Jersey City.

TEXAS CHAPTER

The Texas Chapter of the American College of Chest Physicians held its annual meeting May 5, 1952 on the Roof Garden of the Adolphus Hotel, Dallas. During the luncheon, a business meeting was conducted, and the following officers were elected: President, Robert B. Morrison, Austin; First Vice President, Henry Hoskins, San Antonio; Second Vice President, Howard Smith, Austin; Secretary-Treasurer, Samuel Topperman, Tyler. The scientific program included the following presentations:

"Physiologic Studies in Mitral Stenosis,"
Don W. Chapman, Houston.

"Surgical Treatment of Mitral Stenosis,"
Denton Cooley, Houston.

"Primary Tumors of the Auricle of the Heart,"
W. G. Brown, Houston.

"Tumors of the Thymus Gland,"
Russell Leonard and Fred Aves, Houston.

"Upper Lobe Bronchiectasis,"
John A. Wiggins, Fort Worth.

"Spontaneous Pneumothorax (Idiopathic),"
H. F. Carmen, Dallas.

"Bagasse Disease of the Lungs: Report of Two Cases,"
Shaw McDaniel and John G. Hull, Houston.

X-Ray Conference,
J. E. Miller, Dallas, Moderator.

VIRGINIA CHAPTER

The Virginia Chapter of the American College of Chest Physicians held its annual meeting, Wednesday, May 7, 1952 at the Hotel Roanoke, Roanoke, Virginia. The new officers elected were: President, C. W. Scott, Piedmont Sanatorium, Burkeville; Vice President, W. E. Roye, Veterans Hospital, Richmond; Secretary, W. F. Apperson, State Department of Health, Richmond. The scientific program was as follows:

"Pitfalls of the Surgical Treatment of Bronchiectasis,"
Charles P. Cake, Arlington.

"The Solitary Nodule of the Lung,"
C. Lydon Harrell, Norfolk.

"Pulmonary Blebs and Bullae,"
George A. Welchons, Richmond.

"Extrapleural Pneumothorax,"
William A. Johns, Richmond.

College News Notes

Doctor H. Corwin Hinshaw, San Francisco, California, spoke at a scientific conference preceding the corner stone laying ceremonies at the Institute of Microbiology, Rutgers University, New Brunswick, New Jersey on May 6, 1952.

The conference and dedication were part of a program which included the inauguration of Doctor Lewis Webster Jones, 15th president of the New Jersey State University on May 8. Doctor Hinshaw discussed the place of isonicotinic acid hydrazide in the management and treatment of tuberculosis.

Doctor S. Waxman, recipient of the 1951 Medal of the American College of Chest Physicians also participated in the program.

Dr. Louis Mark, Columbus, Ohio, Past President of the College, recently returned from a tour of Europe and the Near East, where he delivered a series of lectures. Dr. Mark also discussed College activities with the officials of the College in many of the countries visited. Among the countries visited were France, Italy, Greece, Israel, Spain and Portugal. A motion picture film "On the Use of Tryptar" was shown in many of these countries. Dr. Mark was accompanied by Mrs. Mark on this tour.

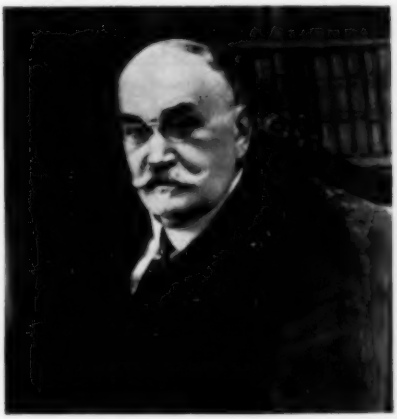
Dr. Paul H. Holinger, Chicago, Illinois, Chairman of the Committee on Motion Pictures, was elected President of the Chicago Tuberculosis Society.

Dr. Joseph S. Hiatt, McCain, North Carolina, was elected District Councilor for the Fifth District of the Medical Society of the State of North Carolina. This state is divided into ten districts and the councilors comprise the Executive Committee. He was also elected Chairman of the Section on Medicine for 1952-1953 for the State Medical Society.

Dr. Andrew Banyai, Milwaukee, Wisconsin, was guest speaker at the staff dinner of St. Joseph's Hospital in Milwaukee on May 13. He spoke on the subject of "Cough." Before the dinner he was entertained at a social gathering by the local members of the American College of Chest Physicians and the alumni members of Marquette Medical School.

Obituaries

GUSTAVE DERSCHEID



In the death of Dr. Gustave Derscheid, Belgium and the entire world has lost one of the greatest physicians, whose name for more than fifty years has been identified with the fight against tuberculosis. In 1897, Dr. Derscheid created the first free clinic for tuberculosis at the Brussels Polyclinic. In 1902, he founded the "Societe Anonyme des Sanatoriums Populaires," which society built two institutions at La Hulpe-waterloo, both of which became famous for the treatment of tuberculosis. He was the organizer of the "Association Nationale Belge Contre La Tuberculose" in 1923, and in 1924, he organized the "Societe Belge d'Etudes Scientifiques." In 1929, Dr. Derscheid founded the "Oeuvre Nationale Belge de Defense Contre La Tuberculose," of which he served as the president until 1950.

These are only a part of the many activities with which Dr. Derscheid was affiliated. His scientific work included 63 publications, many of which were written in cooperation with Dr. Toussaint. He was one of the first physicians in Belgium to use collapse therapy in the treatment of tuberculosis. He was a tireless worker, and neither his age nor the cruel loss of his son, executed by the Germans, could break his spirit. Belgium recognized the high merit of this untireable pioneer by bestowing upon him many honors, among which were Commander of the Order of the Crown, Commander of the Order of Leopold II, Officer of the Order of Leopold and Officer of the Legion of Honour.

Dr. Derscheid belonged to many national and international medical

societies and was a Fellow of the American College of Chest Physicians.

Although in ill health for several years, he was still hard at work when he succumbed at the age of 82. An impressive funeral service took place at Sterrebeek, where a large number of his friends had gathered to pay him a last tribute.

In memory of the esteem in which she held this great man of action, Her Majesty, Queen Elisabeth, Queen of the Belgians, sent her representative to his funeral. The International Union Against Tuberculosis was represented by Professor Bernard. Numerous other officials, physicians, patients, his co-workers and even children escorted the deceased to his last resting place.

Henry Durieu, Governor for Belgium.

LOUIS COHEN
1900-1952

It is with very real sorrow that the passing of Dr. Louis Cohen of Philadelphia is recorded. Dr. Cohen died of a cerebral hemorrhage on February 22, 1952. He was 52 years of age.

Dr. Cohen, a native of Philadelphia, graduated from the Medical School of the University of Pennsylvania in 1923, following which he served one year in postgraduate study in Vienna. He returned to Philadelphia and his advance in his chosen specialty was rapid indeed. Dr. Cohen soon became recognized as a leading authority on pulmonary diseases, and at the time of his death, among his many positions were: Medical Director of Eaglesville Sanatorium, Professor of Clinical Medicine at Temple University Medical School, Assistant Chief in the Tuberculosis Division of Philadelphia's City Department of Health, and Chief of Service in the Tuberculosis Department of the Philadelphia General Hospital. He was Governor of the American College of Chest Physicians for Pennsylvania from 1946 to 1949.

Dr. Cohen was widely known, especially as an excellent teacher and for his broad knowledge of pulmonary diseases. Modest in his manner, friendly in his attitude, and generous of his ability, he had a host of friends and admirers. He will be long remembered and sorely missed by those of us who knew him so well.

Ross K. Childerhose, Governor for Pennsylvania.

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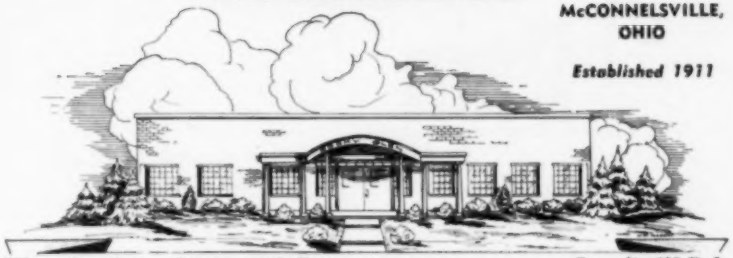

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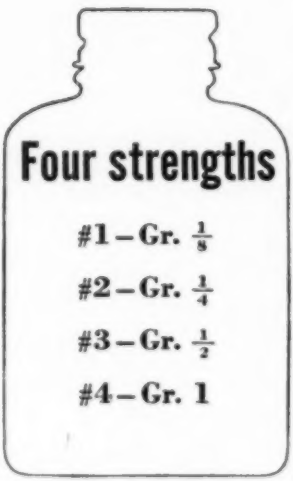

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Medical Director wanted for tuberculosis hospital at Fall River, Massachusetts. Must have Massachusetts license or one reciprocal with Massachusetts. Salary $6700 per year and maintenance. Write direct to Dr. Arthur J. Ledoux, Health Commissioner, Fall River General Hospital, Stanley Street, Fall River, Mass.

Resident physician wanted for active thoracic surgery service. Salary commensurate with experience. Includes maintenance. Tuberculosis hospital. Write directly to Dr. Nathan Levene, Hazelwood Sanatorium, Bluegrass Avenue, Louisville, Kentucky.

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NEWS NOTES

The following officers were elected by the Society of Tuberculosis of Rosario, Argentina: President, Abraham F. Schottlender; General Secretary, Luis Cabanillas; Acting Secretary, Raul Tisserandet; Treasurer, Domingo Pacheco; Directors, Isaac Alberto Hassan, Juan J. Boretti and Ernesto Vigetti.

The newly elected officials of the Sociedad Argentina de Tisiologia for the year 1952 are as follows: President, Augusto Casanegra; Vice President, Abel Cetrangelo; Secretary, Horacio Rodriguez Castells; Treasurer, Jorge Tamini.

The First Congress of the Universities of the Argentine Republic will be held at Cordoba in October 1952, celebrating the 75th anniversary of the Foundation of the Faculty of Medical Sciences.

The Chilean Society for Tuberculosis, meeting at Santiago, elected the following officers for 1952-1953: President, Sotero del Rio Gundian; Secretary, Oscar Botteselle Pahul; Treasurer, Eduwardo Calderon Paul; Directors, Hernan Oyanguren Moya and Miguel Etchebarne Riol.

The problem of tuberculosis among persons who move from one place to another to make their living—migratory farm workers, for instance—demands sober consideration. In most states a means test is still one of the standards of eligibility for sanatorium care—a practice which shows failure to recognize fully not only the communicability of tuberculosis but also the social and economic impact of the disease. Robert J. Anderson, M.D., Medical Papers of the Annual Meeting of the Canadian Tuberculosis Association, May, 1951.

Tuberculosis is a personal tragedy and a community problem. It begins with the individual and must be fought in the individual and in his environment. Will Ross, former president of the NTA.

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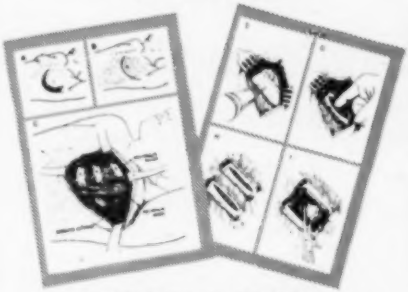

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Chicago Postgraduate Course, October 6-10, 1952.

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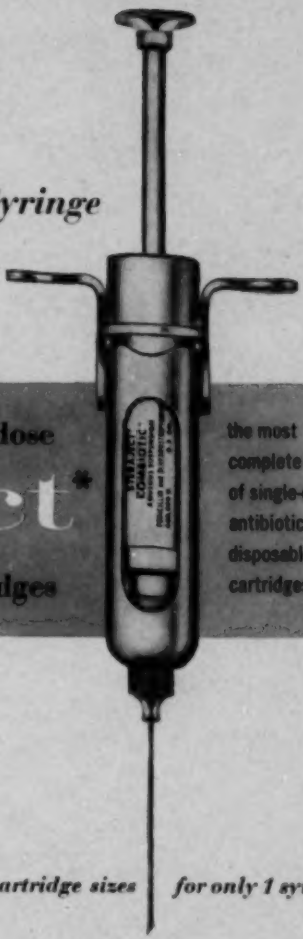

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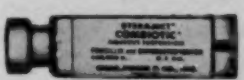

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